THE BRITISH VETERINARY JOURNAL

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EDITORIALS

ROYAL LEAMINGTON SPA

WHAT could be better than that a President from the "Midlands" should call his men to their annual conclave and carousal in the heart of England and at Royal Leamington Spa? We pass by the thought lightly he might have surmised, that clear heads and quick wits are a necessary adjunct to scientific discussion, and that the medicinal waters would play their part in dispelling the aurors consequent upon the social activities of the "night before."

Leamington is old and visitors will be shown, and with pride, an oak tree, the Midland Oak, which is said to be 900 years old and is reputed to mark the centre of England. For centuries it was a village and of no great consequence until in 1784 the medicinal qualities of certain natural springs were brought to a relatively wide public notice. That date marks the birth of the modern town and the seal was placed on its renown when Queen Victoria and her mother, the Duchess of Kent, paid a visit in 1838 and thereafter it became known as Royal Leamington Spa.

Leamington sits placidly on the River Leam near the latter's junction with the Avon and in a countryside which is famed for its beauty and for hunting and other field sports. Its proximity to Stratford on Avon, Warwick and Kenilworth gives added attraction to those of literary or historical bent. The old Parish

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Church speaks of its age, the Regency architecture, the Pump Room gardens, Jephson gardens and Victoria Park tell of its growth to fame in more recent years. Scholars will remember that its High School for Girls is famed throughout the English-speaking world.

Leamington's renown rests mainly on its fame as a Spa, but it is an industrial centre also. The latter has to do with iron and steel foundry work and is employed in the manufacture of stoves, electric vehicles and component parts for motor cars. Leamington is a place in which the old and the new have been combined to save the town's fair face and yet bring that vigour which is essential to progress. All the signs are set fair for a good congress, the scientific and social programmes are full of interest and pleasure and the Borough is renowned for its hospitality.

SHEEP

There was a time when sheep roamed and waxed fat in England and wool growing was a major factor in British agronomy. The great churches, now seemingly out of all proportion to the place in which they stand, are a constant reminder of the part that sheep and wool played in the economy of the countryside. Alas! these days are gone and the sheep have been demoted to a subsidiary role on our farms. None the less the sheep are still a valuable asset and especially so at the present time when, because of the ravages of two world wars, we have to make the best possible use of our own resources. Sheep provide us with food and clothes but these things are not an adequate measure of their value to the farmer. They are one of the best means of preserving and enhancing the fertility of the soil. Some light land areas could not be kept in economic balance but for sheep. In Britain also, through good times and bad, sheep husbandry has been carried on at a high level and the various indigenous breeds have been developed bearing in mind their varied propensities for the production of meat and wool at the best economic level.

Foreigners have not been slow to recognise their qualities and they are accustomed to come to Britain to re-stock and up-grade their flocks. Like our horses and cattle our sheep seldom fail in this respect. So another factor has been added to the value of sheep to the British farmer, the export trade, and in some cases the economic return outdistances that to be derived from meat and wool. Veterinary surgeons are called to attend to sheep more frequently than was the case in the past, for under the pressure of modern conditions even individual sheep are of considerable value and veterinary help and advice can pay good dividends to the farmer.

THE BRITISH VETERINARY JOURNAL is pleased to publish articles which deal with the management of sheep in health or disease.

BRITISH VETERINARY ASSOCIATION

ROYAL LEAMINGTON SPA CONFERENCE, 1956

PROGRAMME

(All meetings will be held in the Great Hall of the Town Hall unless otherwise stated.)

SUNDAY, SEPTEMBER 16

6.30 p.m.—Service at All Saints' Parish Church, Bath Street, Leamington, conducted by the Vicar—Rev. Canon R. C. Streatfield, M.A.

8.00 p.m.—"At Home" given by the Midland Counties and East Midlands Divisions, at the Regent Hotel, Leamington.

MONDAY, SEPTEMBER 17

10.00 a.m.—Blessing: Rev. Canon Streatfield, M.A. Opening of the Congress by His Grace The Duke of Devonshire, M.C. Civic Welcome by the Mayor of Royal Leamington Spa.

10.30 a.m.—Ladies' excursion: Visit to Cheltenham, including Broadway and the Slaughters.

10.45 a.m.—Opening of Veterinary Exhibition by the President, B.V.A.

12.00 noon to 1.00 p.m.—Films.

2.30 p.m.—*Paper:* "Review of Present Knowledge of Johne's Disease." *Author:* Mr. T. M. Doyle, F.R.C.V.S., D.V.S.M. *Openers:* Mr. J. B. White, M.R.C.V.S., and Mr. J. D. Rankin, M.R.C.V.S. *Chairman:* Mr. A. L. F. Mullen, M.R.C.V.S., D.V.S.M. *Recording Secretary:* Mr. D. C. Croft, B.V.SC., M.R.C.V.S.

8.30 p.m. to 1.00 a.m.—Civic Reception and Dance at the Town Hall, by kind invitation of the Mayor and Corporation of Royal Leamington Spa.

TUESDAY, SEPTEMBER 18

9.30 a.m.—*Paper:* "Farm Health Problems: Where Does Britain Stand To-day?" *Author:* Mr. S. L. Hignett, B.SC., M.R.C.V.S. *Opener:* Sir James Scott Watson, C.B.E., M.C. *Chairman:* Mr. E. R. Callender, O.B.E., M.R.C.V.S. *Recording Secretary:* Miss W. M. Brancker, M.R.C.V.S.

10.30 a.m.—Ladies' Guild Annual General Meeting.

2.00 p.m.—Demonstrations by close-circuit Television at the Moreton Morrell Institute, Nr. Leamington (by kind permission of the Principal).

6.00 p.m.—Alumnus Dinners.

8.30 p.m.—Ladies' Guild Dance at the Spa Ballroom, Leamington.

WEDNESDAY, SEPTEMBER 19

9.30 a.m.—*Paper:* "Present-day Toxicity Problems: A Review." *Author:* Mr. J. L. McGirr, B.SC., M.R.C.V.S. *Openers:* Mr. A. Steele-Bodger, M.A., B.SC., M.R.C.V.S., and Mr. N. M. Black, M.R.C.V.S. *Chairman:* Mr. W. L. Dwerryhouse, M.R.C.V.S., D.V.S.M. *Recording Secretary:* Mr. T. C. Denholm, M.R.C.V.S.

10.30 a.m.—Ladies excursion: Visit to Kenilworth, Guys Cliffe and Stoneleigh.

11.30 a.m.—*Paper: In the Small Hall*—"Common Disease of Cage Birds and Other Less Usual Pets." *Author:* Mr. O. G. Jones, M.R.C.V.S. *Opener:* Mr. I. F. Keymer, M.R.C.V.S. *Chairman:* Mr. S. Jennings, M.R.C.V.S. *Recording Secretary:* Mr. G. V. Laugier, M.R.C.V.S.

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12.30 p.m.—Last Meeting of Council, 1955/56.

2.30 p.m.—Congress Photograph.

3.00 p.m.—Annual General Meeting, with Presidential Address.

7.30 for 8.00 p.m.—Annual Banquet at the Leofric Hotel, Coventry.

THURSDAY, SEPTEMBER 20

9.30 a.m.—*Paper:* "The Anatomy of Disc Protrusion in the Dog." *Author:* Dr. A. S. King, B.SC., M.R.C.V.S. *Openers:* Mr. S. W. Douglas, M.A., M.R.C.V.S., and Dr. G. B. Brook, F.R.C.V.S., F.R.S.E. *Chairman:* Mr. J. S. Steward, F.R.C.V.S., D.V.S.M. *Recording Secretary:* Mr. W. H. Carr, B.V.SC., M.R.C.V.S.

9.30 a.m.—*Paper: In the Small Hall*—"Artificial Insemination in Pigs." *Joint Authors:* Mr. C. Polge, B.SC., and Mr. L. E. A. Rowson, O.B.E., M.R.C.V.S., *Openers:* Mr. D. H. L. Madden, M.R.C.V.S., and Mr. A. F. Holt, B.SC., M.R.C.V.S. *Chairman:* Mr. W. P. Stokes, M.R.C.V.S. *Recording Secretary:* Mr. M. Steele-Bodger, M.R.C.V.S.

11.30 a.m.—*Paper: In the Small Hall*—"Foot-rot in Sheep. *Author:* Professor W. I. B. Beveridge, D.V.SC.(SYD.), M.A. *Opener:* Miss A. I. Littlejohn, B.V.SC., M.R.C.V.S. *Chairman:* Mr. J. G. Murray, M.R.C.V.S., DIP.BACT. *Recording Secretary:* Mr. H. E. Roberts, B.SC., M.R.C.V.S.

2.00 to 4.00 p.m.—Special Film Programme. Golf, Tennis, Bowls, etc.

3.00 p.m.—Mannequin Parade at the Lord Leycester Hotel, Warwick (for the ladies).

7.30 p.m.—Free, or visit to Shakespeare Memorial Theatre to see "Love's Labour's Lost." (*Application for tickets should be made immediately to B.V.A. Headquarters.*) (For those attending this performance arrangements are being made for dinner to be served at 6.30 p.m. at the Red Horse Hotel, Stratford-on-Avon.)

FRIDAY, SEPTEMBER 21

9.30 a.m.—*Paper:* "Animal Diseases from the Point of View of Human Medicine." *Joint Authors:* Dr. P. G. Arblaster, M.R.C.P., and Dr. A. P. Prior, B.S. *Openers:* Dr. N. S. Barron, B.SC., M.R.C.V.S., and Mr. H. T. Matthews, M.A., B.V.SC., F.R.C.V.S., D.V.H. *Chairman:* Mr. E. R. Corrigall, M.R.C.V.S., D.V.S.M. *Recording Secretary:* Mr. A. F. Ritchie, M.R.C.V.S.

11.30 a.m.—*Paper: In the Small Hall*—"Examination of the Eye with Special Reference to the Horse and Dog." *Author:* Professor C. Formston, F.R.C.V.S. *Opener:* Mr. S. F. J. Hodgman, M.R.C.V.S. *Chairman:* Major C. Chambers, O.B.E., F.R.C.V.S. *Recording Secretary:* Mr. G. Eaton, F.R.C.V.S.

2.30 p.m.—Ladies' excursion: Visit to Compton Wynyates and Ettington Park.

2.30 p.m.—*Paper:* "Poultry Breeding for Increased Production with Special Reference to Health." *Author:* Mr. J. O. L. King, M.V.SC., B.SC., M.R.C.V.S. *Openers:* Mr. G. G. Belfield (Sterling Poultry Products, Ltd., Salisbury) and Miss E. E. Kidd (President, Chick Producers' Association, London). *Chairman:* Mr. E. G. Morris, M.R.C.V.S. *Recording Secretary:* Mr. A. H. Hogg, M.R.C.V.S.

2.30 p.m.—Films.

8.30 p.m. to 1.00 a.m.—President's Reception and Dance at Chesford Grange.

SATURDAY, SEPTEMBER 22

10.00 a.m.—Closing Meeting of Congress. First Meeting of Council, 1956/57.

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GENERAL ARTICLES

AN HISTORICAL NOTE ON THE DISCOVERY OF THE DEPRESSOR NERVE

By A. S. KING

Department of Veterinary Anatomy, The University, Bristol, 8

CERTAIN events of a personal nature at the time of the discovery of the depressor nerve by Cyon and Ludwig in 1866 were described by the distinguished physiologist, the late Alfred Schweitzer, in a public lecture in 1949. Coming from such an authoritative source, this account rightly gains considerable credence. But in justice to the historic figures whose reputations are involved we should not accept without some inquiry an essentially hearsay account of events of nearly one hundred years ago. All those who worked with Cyon and Ludwig must long since be dead, and therefore we have no "witnesses of fact"; in any case, like most murders, the principal act was probably witnessed only by the two protagonists. The problem is therefore now an historical one. The following note outlines the story as told by Schweitzer and then tests it against the characters and personal circumstances of the two men as revealed by their writings and contemporary comment.

In the year 1865 Kark Friedrick Ludwig and Elie Cyon entered the Leipzig Institute of Physiology, the former as Director and the latter as a junior research assistant. According to Schweitzer's account Ludwig soon became dissatisfied with Cyon's work, reprimanded him, and demanded an immediate research contribution on pain of speedy dismissal. Hastily turning to the rabbit, Cyon put electrodes indiscriminately under the various cervical nerves: one of these happened to be the depressor. Thus, we are told, one of the outstanding physiological discoveries of the century was made, not as a planned investigation but as an inconvenient improvisation. In a moment Cyon's position was transformed from possible disgrace to certain fame . . . "so thin is the line which divides success from failure." Just how remarkable the discovery was can be measured from the fact that it preceded by over half a century Hering's discovery of the function of the carotid sinus nerve: yet throughout the mammalian orders the carotid sinus nerve is anatomically so much more conspicuous than the depressor.

Hirsch's Lexicon of 1888 provides some biographical details of both Ludwig and Cyon. Ludwig, a German, had held chairs before, in Zurich and Vienna. But his appointment in 1865, at the age of forty-nine, to the Directorship of the Leipzig Institute marked the summit of his career. In the years that followed he sponsored a remarkable range of researches and became widely recognised as one of the most successful research organisers of the nineteenth century.

Cyon was born in Russia in 1843. After studying in the medical schools of Warsaw, Kiev and Berlin, he graduated at the latter in 1864 and again at

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St. Petersburg in 1865. Hirsch next speaks of him as Dozent for Anatomy and Physiology at St. Petersburg in 1868. There is a gap in Hirsch's biography from the date of graduation in 1865 to the appointment as Dozent in St. Petersburg in 1868. But luckily Cyon's movements during this important period can be reconstructed from his articles of 1865, 1866 and 1867. In the first article which he published, in 1865—an experimental study of the dorsal and ventral roots of the spinal cord—he mentioned that the work had been done in the Leipzig Institute. In 1866 he and Ludwig jointly presented the discovery of the depressor at the November session of the Royal Medical Society of Saxony ; it is certain that this work was done also at Leipzig because a facsimile was published in 1867 in the Report of the Leipzig Institute of that year. By the end of 1866 Cyon had left Leipzig and was working under Professor Du Bois-Reymond (then Professor of Physiology in Berlin University) : this fact transpires from acknowledgments made in an article on the innervation of the heart, published jointly with his brother, M. Cyon, in 1866. A similar acknowledgment in a paper published the following year shows that he was still with Du Bois-Reymond in 1867. Thus it is established that Cyon joined the Leipzig Institute in 1865 and left in 1866 to go to Berlin University where he remained until 1867.

After this his rise to fame was meteoric. In 1870 he won the gold medal of the Paris Academy, and two years later at the age of only twenty-nine was appointed to the Chair of Anatomy and Physiology at St. Petersburg. In the same year he took up politics and gained the confidence of the Tsar, who commissioned him to eliminate nihilistic intrigue from the St. Petersburg Academy. Five years later his enemies forced his resignation but the Emperor remained his patron, appointing him to the Privy Council and raising him to the aristocracy. Soon afterwards he accepted an invitation from Claude Bernard to go to Paris, which subsequently became his headquarters. There were seven books among which were treatises on politics and philosophy. His scientific work included outstanding investigations into the functions of the nervus accelerans and splanchnic nerve, the method of fat formation in the liver, and the rate of propagation of nerve impulses in the spinal cord. There were other articles on a wide range of subjects; for example, on the sense organs, temperature regulation, and many aspects of circulatory, respiratory and nervous physiology. Incidentally, he noticed and accurately drew the carotid sinus nerve of the horse, and declared his intention of investigating it experimentally': "Nicht minder interessant ist das Nervennetz, welches vom Ganglion cervicale supremum enstammt und, die Art. carotis umgriefend, in die Wandung deselben eintritt, und zwar meistens in der Richtung nach unten. Auch diese Nerven sollen nächstens einer experimentallen Prüfung unterworfen werden." Had he carried out this intention he would in all probability have deprived Hering of the renown of 1924.

Clearly Cyon, like Ludwig, was a very distinguished scientist. But, unlike Ludwig's, Cyon's work had flaws which become apparent on closer inspection (see for, for example, his series of articles on the mammalian depressor nerve). He tended to ignore or summarily dismiss work by others, to be

casual in describing important techniques, and to draw dramatic conclusions from isolated evidence . . . indicative of obstinacy and pride, of a disregard for the systematic method, and of a surprisingly undeveloped critical faculty.

So from their careers and written work the characters of the two men emerge. Ludwig was a man of responsibility and solid accomplishment, methodical and reliable: Cyon was an individualist, spectacular and inconsistent . . . a brilliant showman. Two men of similar scientific distinction and more contrasting character can hardly be imagined.

What is the evidence for and against the story of the discovery of the aortic nerve? First, is there anything to justify the charge of idleness, The only evidence is that Cyon published one article in 1865. This was by no means an insignificant work; at the least it shows that he had been active during 1865. It could even be argued that such a work was a distinct accomplishment for the first year of research. Certainly there could be no reason for a reprimand on grounds of idleness during this period. On the other hand, until November of 1866, when the discovery of the aortic nerve was reported, this was the only work which Cyon had produced; there is no evidence that he was doing anything during the first half of 1866. By Cyon's subsequent standards this was undoubtedly an unproductive phase: in 1867, 1868, 1869 and 1870 he published an average of three papers each year, and a book as well.

To what extent do Cyon's movements throw light on the problem? It is a fact that he departed from Leipzig immediately after the discovery of the aortic nerve. It is surprising that Ludwig allowed his junior to go so quickly after such a momentous discovery. In normal circumstances surely the Director would try to retain and develop the services of such a promising assistant. This hasty move to Berlin conforms much more closely to the idea of strained personal relationships.

The complete gap in Hirsch's biography between 1865 and 1868 is strange. Presumably Cyon himself supplied these details of his career. If the story of the discovery is true, Cyon's part in it was somewhat undignified; also Ludwig's reprimand would have been a lasting blow to his pride. Therefore there are adequate motives for Cyon to omit his sojourn at Leipzig from the records.

To what extent do the careers and personalities of the two men assist our judgment? Both were appointed at Leipzig in the same year. Ludwig, a man with a growing reputation for the initiation and supervision of research, had just achieved one of the most important posts attainable. What could be more natural than emphatic and insistent demands on his staff? On the other hand Cyon, as a recent graduate, was at that moment making the adjustment from student to researcher . . . a transition which is not always smooth and easy.

Finally, if the reprimand really was given, can we believe in such an astonishing result? Cyon's subsequent career, considered in terms of both his scientific and political enterprises, was essentially uneven, being punctuated alternately by glories and catastrophes; perhaps he had genius for genius is often

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unbalanced. The discovery on the spur of the moment of something so extraordinarily important as the depressor nerve was typical.

The relatively small output of work while at Leipzig, the hasty move to Berlin, the gap in Hirsch's biography, the clash between Ludwig's career and Cyon's transition from student to scientist, and the evidence of Cyon's temperament, conform to Schweitzer's account. We shall never know for certain what happened, for all this evidence is purely circumstantial. However, since there is seldom smoke without a fire and since there is evidence of a combustible mixture of careers, circumstances and personalities, I think a reasonable man can accept the romantic story of the great discovery.

Acknowledgment

It is a pleasure to thank Professor E. C. Amoroso for his interest and advice.

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STUDIES ON THE CLIMATE OF PIG HOUSES

II—Air Temperature and Humidity in Piggeries

By D. W. B. SAINSBURY, Ph.D., B.Sc., M.R.C.V.S., and
N. J. SCORGIE, M.A., B.Sc., M.R.C.V.S.
Department of Veterinary Hygiene, Royal Veterinary College, London, N.W.1

In the first part of these studies (Sainsbury and Scorgie, 1955), attention was drawn to the difficulty of maintaining a satisfactory climatic environment in pig houses at all times of the year. It was also pointed out that since the pig is physiologically ill-adapted to climatic extremes, cold, damp, draughty and fluctuating conditions must be avoided if pigs are to be reared satisfactorily. We discussed the ventilation of pig houses, pointing out the many faults in existing systems and described methods which could be adopted to secure a satisfactory air flow and ventilation rate for all types of weather likely to be met with in the British Isles.

In this second part of the studies, further data obtained from our survey are given, relating in this case to the air temperature and humidity status in piggeries.

Air Temperature

Heat losses from piggeries. In our previous publication we observed that faulty ventilation could be responsible for the dissipation of a large amount of the heat radiated by the pigs, which would otherwise be utilised to warm the

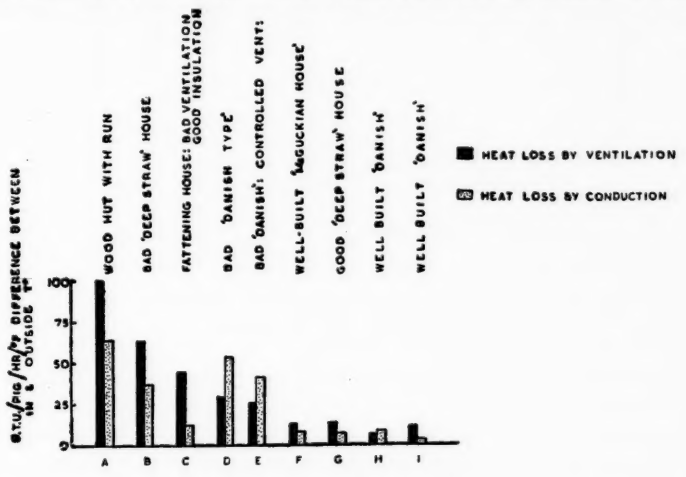


FIG 1 HEAT LOSSES FROM NINE DIFFERENT TYPES OF PIG HOUSES

building. The other main cause of heat loss from a piggery occurs by conduction from the inside air to the outside atmosphere through the building's surfaces.

In Fig. I are shown the calculated heat losses (B.T.U.) per pig per hour for each 1° F. difference between the inside and outside air temperatures for

nine piggeries, designated A to I respectively, representing most types of house found in practice. The considerable difference between the losses for well-built houses on the one hand and, on the other, those that are badly insulated and/or badly ventilated, is readily apparent.

These data assume practical importance when considered in relation to the "sensible" heat production of a fattening pig of known weight. A pig of 100 lb. liveweight produces approximately 450 B.T.U. of "sensible" heat per hour under average winter conditions (Mitchell and Kelley. 1938; Bond, Kelly and

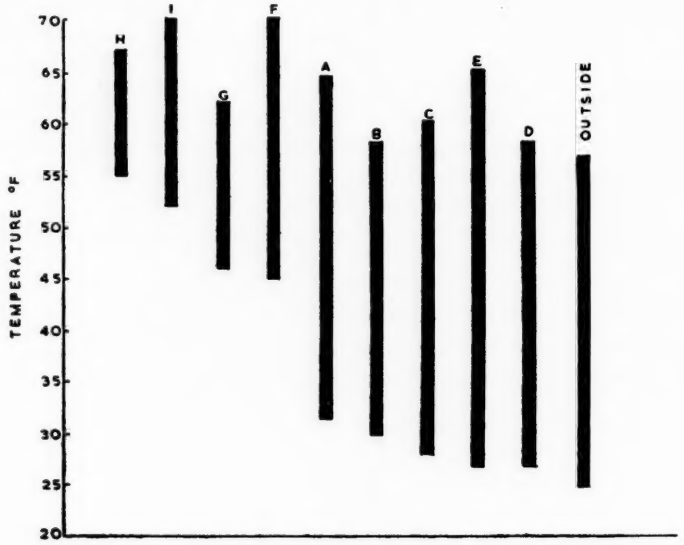
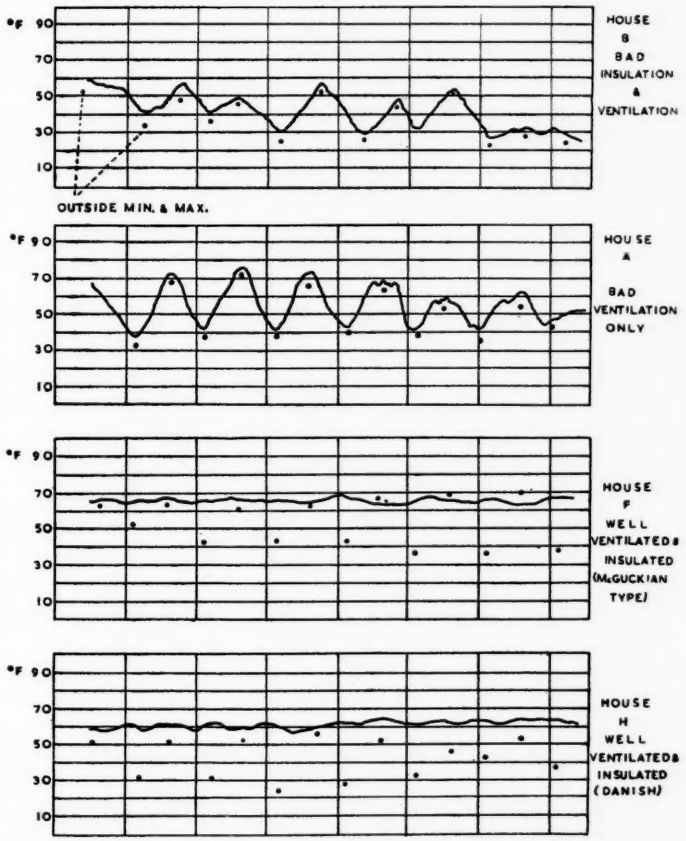


FIG II EXTREME TEMPERATURE VARIATION WITHIN EACH OF NINE PIGGERIES, & OUTSIDE, FROM 1 DEC 52 TO 28 FEB 53

Heitman, 1952). If this is compared with the heat lost through ventilation and surface conduction it will be found that the temperature inside a badly constructed house can be at best only 7.5° F. above that outside and, at the worst will be only 3° F. On the other hand, the differences in air temperature between inside and outside in the case of well-constructed houses may be as great as 20° F., and even 30° F. or more on occasion. These figures are based on the theoretical heat loss by conduction through the surfaces, together with the heat lost by ventilation, which is derived from our own work on determination of ventilation rates using Haldane's portable apparatus for the measurement of the CO_2 content of the air. That these estimated heat losses were borne out in practice can be seen by studying Fig. II, which shows the extremes of temperature from December, 1952, to February, 1953, in the same piggeries which were the source of data for Fig. I. The extremes of outside temperature for the relevant period are given for comparison. It was found by observation that differences in the minimum air temperatures between inside and outside

throughout the period December to January were from 20° F. to 27° F. in the well-built houses (excluding the mechanically heated and ventilated piggery), and from 2° F. to 8° F. in other types of house. These figures show a remarkably close correspondence to the calculated estimates given in the preceding paragraph.

FIG III COPIES OF THERMOGRAMS (WEEKLY) TAKEN IN 4 PIGGERIES

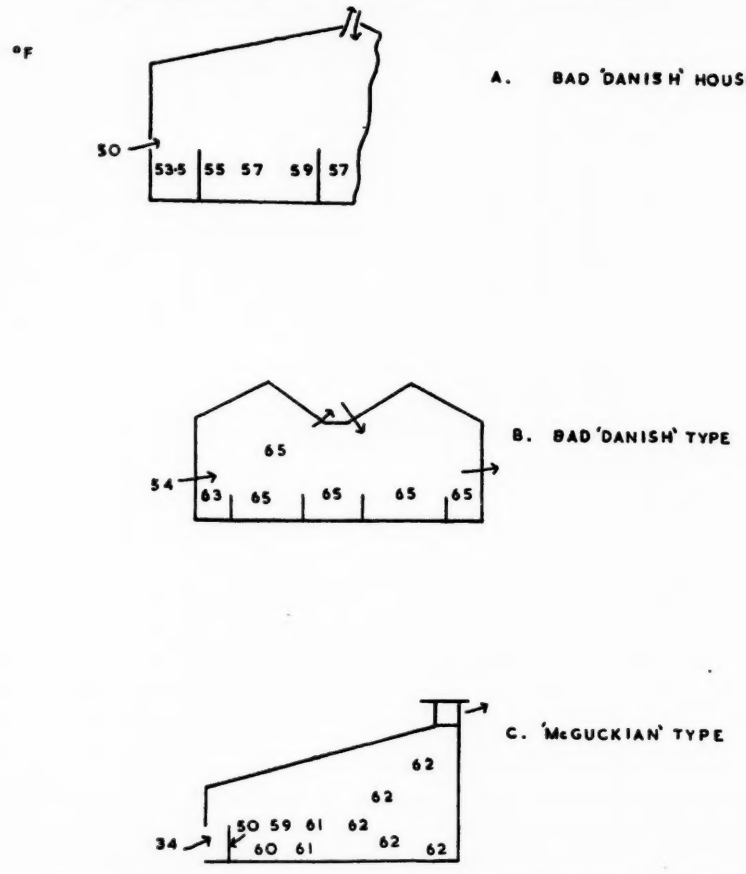


Temperature variation in the piggeries. It follows from the facts we have given in regard to heat losses that only in piggeries which are both adequately insulated against heat loss via the surfaces and have fully controllable ventilating systems, can a large temperature differential between inside and outside be maintained. An additional advantage gained by having good ventilation and thermal insulation is that the diurnal temperature variation for inside air can be reduced to a very low value, even to zero, despite wide fluctuations in outdoor temperatures. This is in marked contrast to houses having deficiencies of either ventilation or thermal insulation, or both, in which the temperature levels closely follow those of the outside air. Four copies of thermograms (Fig. III) recorded over

weekly periods show these points well. Minimum and maximum outside temperatures are marked at the appropriate points on the thermograms.

Thermograms for houses A and B exemplify the state of affairs likely to exist in badly ventilated or poorly insulated houses, whilst houses F and H illustrate the remarkable stability of temperature possible in a well-designed and constructed piggery.

Fig. 4
Examples of temperature gradients in three different piggeries.



The temperature gradient within a piggery. Important information on the over-all temperature status of a piggery can be derived from a study of the variation in temperature recorded at different sites in the house. Some variation in temperature throughout a piggery is inevitable when there is a considerable difference between inside and outside temperatures, since the colder fresh air coming in through the inlets will tend to produce greater cooling of the inside air in the region of its entry. For this reason potentially great variations are

possible in well-built houses when there is a considerable difference between inside and outside temperatures.

Surveys of temperature conditions in houses we have examined may be summarised as follows:—

(i) In lofty or cold piggeries typified by the badly-constructed "Danish" houses (Fig. IV, A and B) only small differences of temperature were recorded at different places in the building. The coldest areas were generally in the dunging passages, in the vicinity of the outer walls, and on the windward side.

(ii) Houses with inlet ventilators set high up in the walls and adequately baffled (see Fig. V) showed greater differences in the temperatures at various sites, but since cold draughts did not blow directly into the pen, any temperature gradient that existed tended to be favourable for the pigs—that is, the highest temperatures were recorded in the pens and the lowest at roof level near the inlets.

(iii) Houses with uncontrollable and unbaffled inlets (Fig. VI) had small gradients of temperature. The coldest regions were found to be in the pens along the windward wall and in the centre passage.

(iv) A fairly steep temperature gradient existed in houses with outside yards. In a McGuckian type piggery (Fig. IV, C), the lowest temperatures, as might be expected, were just inside the baffled doorway between the pen and the run. The hanging of a sacking curtain greatly reduced the gradients and the recordings showed that in the case of a well-managed McGuckian house during the winter months the temperature gradient from 1 foot away from a baffled door to the chimney outlet varied by no more than 4° F.

Humidity

(*a*) *Relative humidity.* Comparison of records of relative humidities during winter in different types of piggeries studied revealed there were marked differences between them. In houses well ventilated and properly thermally insulated the relative humidity averaged approximately 70 per cent, a house with mechanical ventilation and artificial heating having, not surprisingly, the most constant values, never going over 80 per cent and generally being considerably below. "Peaks" of humidity in this piggery occurred at feeding times; the lowest values were recorded during the night, presumably due to the inactivity of the pigs at this time coupled with the more constant air flow rate. In a good "McGuckian" house (well insulated pens with partly covered yards attached) relative humidities were again generally below 70 per cent, "peaks" occurring at feeding times but rapidly falling afterwards to their original value; the lowest relative humidities were again found during the night. It is worthy of note that in the McGuckian house this low humidity level was achieved without either any mechanical aid to ventilation or any means of artificial heating. The relative humidity picture in a well-built Danish-type house was similar, though here the relative humidity was generally higher during the night than during the day, this being explained by the slight fall in night temperature and an actual increase in air humidity from accumulated dung and urine. The only Danish-type house

Fig. 5
Typical temperature gradients in piggeries with baffled inlet ventilators.

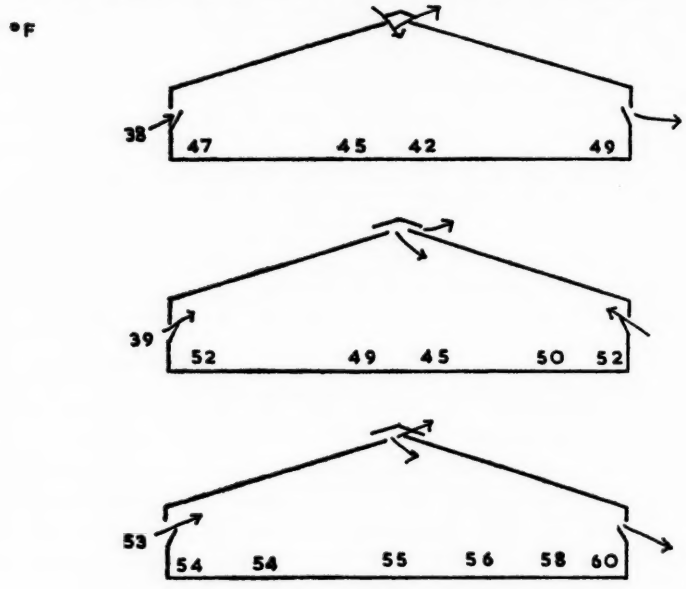
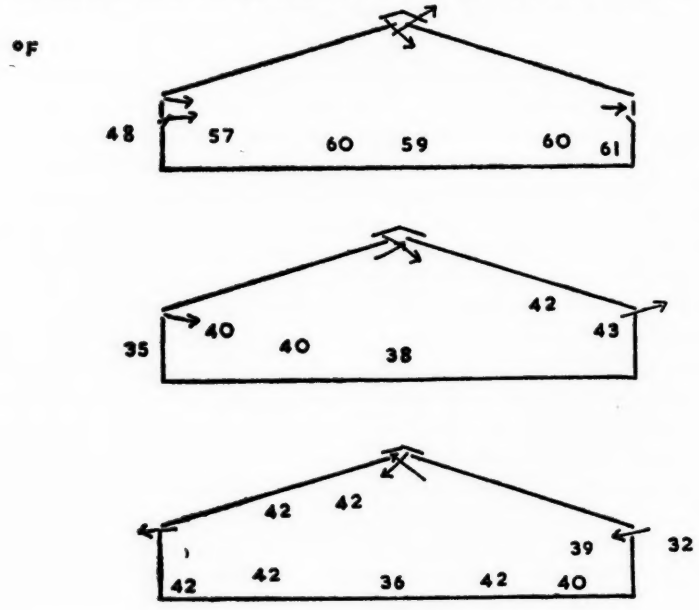


Fig. 6
Examples of temperature gradients in houses with poorly-baffled inlet vents.



in which the relative humidity was lowest during the night was a building where the dunging passages were separated from the pens by walls carried up to the ceiling, a small low entrance-way for the pigs being the only connection between the dunging passages and the house as a whole.

Relative humidities in well-constructed "deep-dunging" houses were generally somewhat higher than in houses of other types having comparable construction. A higher air humidity is obviously promoted by the water vapour rising from the deep-strawed area.

In badly ventilated houses, with no thermal insulation of the surfaces, relative humidities were consistently higher than in well-designed houses, and were accompanied by condensation on the roof and walls and a general impression of dampness. In general the temperatures inside these buildings closely paralleled those outside, and the usual diurnal variation of temperature was likewise coincident with diurnal variation of relative humidity. The highest relative humidities, therefore, occurred during the night due, it could be reasoned, not only to the low night temperatures but aided by the diurnal fall in wind speed, and, hence in ventilation rate, and by the gradual "build-up" of excretal pollution through the night.

Winter relative humidities in badly-built houses were usually 80 per cent and above. There was an indication in these houses also of the occurrence of "peaks" of humidity at feeding times but, in contrast to the position in well-constructed houses, former levels of humidity were restored slowly—an indication of the less effective ventilation.

(*b*) *"Absolute" humidity.* Table I gives a summary of the general humidity picture in a representative selection of piggeries during a typical week in winter. It will be seen that the warm, well-insulated and properly ventilated houses had the lowest *relative* humidities, but the highest *absolute* humidities (i.e., actual water content of the air).

Conversely the damp, cold, badly ventilated and uninsulated houses had the highest *relative* humidities but lowest absolute humidities. This latter group of houses had far greater ventilation rates than the former, but this would be essential for even partial removal of the water vapour from the building, owing to the fact that the air passing out of these houses would be but little warmer than the outside air, and would therefore hold only slightly more vapour than it contained when it entered.

It was found that the amount of condensation on surfaces was not clearly relatable to the relative or absolute humidity values of the air, but would appear rather to depend on the efficiency of insulation at the surface, the permeability or otherwise of the material to water vapour, and the adequacy of air flow over the surface. In houses with poor insulation but with some measure of controlled and well-conceived ventilation, condensation was less profuse than in piggeries having both bad insulation and bad ventilation.

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TABLE I

HUMIDITY STATUS IN NINE PIGGERIES DURING THE WEEK ENDING 6.12.52.

PIGGERY	AVERAGE TEMPERATURE °F	AVERAGE RELATIVE HUMIDITY	ACTUAL WATER CONTENT OF THE AIR (grs. moisture per lb. dry air)	CONDENSATION	THERMAL INSULATION	REMARKS INCLUDING ESTIMATED VENTILATION RATES
House "G" (Well constructed deep-straw)	60	76	60	Very slight	Good	800 cu ft/pig/hr. Ventilation well managed.
House "F" (McGuckian type)	60	70	50	Very slight	Very good	400 cu ft/pig/hr. Ventilation very carefully controlled.
House "I" (Well constructed Danish type)	58	60	45	Very slight	Very good	700 cu ft/pig/hr. Ventilation mechanically assisted.
House "H" (Artificially heated and ventilated Danish type)	58	68	50	Very slight	Very good	650 cu ft/pig/hr.
House "D" (Badly built Danish type)	47	84	40	Very severe	Bad	900 cu ft/pig/hr. No ridge ventilation.
House "C" (Well insulated. Badly ventilated)	46	80	37	Severe	Good	1100 cu ft/pig/hr. No control of ventilation.
House "B" (Badly constructed deep-straw)	43	80	33	Severe	Bad	1200 cu ft/pig/hr. Insufficient control of ventilation.
House "A" (Wooden hut with run)	43	85	35	Absent	Average	1750 cu ft/pig/hr. Ventilation excessive & uncontrollable.
House "E" (Bad Danish type)	42	86	34	Very severe	Bad	1000 cu ft/pig/hr. Ventilation uncontrollable & badly planned.

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Summary and Conclusions

1. *Air temperatures.*

Data obtained in the course of a survey of pig houses have demonstrated that in houses with thermally insulated surfaces and controllable ventilation, it is feasible for the air temperatures inside to be maintained at from 20 to 35° F. above those outside. Examples of actual indoor winter temperatures within the range 50 to 65° F. are given. Moreover, diurnal variation of temperature in well-constructed piggeries can be practically eliminated, and notwithstanding the large difference existing between inside and outside temperatures, there need be no great variation of temperature from part to part of a piggery, provided there is proper baffling of ventilators or any other inlet of fresh air. Efficient thermal insulation of surfaces is essential, not only for maintenance of warmth but also to eliminate condensation.

In houses deficient in either ventilation or thermal insulation, or both, no appreciable control over temperatures is possible, and consequently in the cold (winter) and damp atmosphere of this type of house, surface condensation is inevitable.

2. *Humidity.*

It was possible to show real differences between the humidity status in piggeries of good and bad construction. Surface condensation and high relative humidities can be avoided in most instances in well-ventilated and thermally insulated buildings. In other types of piggeries it was impossible to exercise any effective control of the humidity of the air or surface condensation.

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THE PERIDUCTAL LOBULES OF THE BOVINE MAMMARY GLAND

By P. S. BLACKBURN, Ph.D., M.R.C.V.S.
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ACCESSORY glands in the walls of the teat cistern and the gland cistern of the bovine mammary gland have been recognised for a long time, and descriptions of these structures published early in the century are referred to by Turner (1952). More recently Venzke (1940) made a histological study of the teat and gland cisterns of the bovine mammary gland and described the accessory glands in the walls of the cisterns. He considered that as the secretion of these glands appeared to be similar to that of the mammary gland in that secretory granules and fat droplets could be observed, it seemed reasonable to assume that these glands secrete milk. Pattison (1952), describing experimental streptococcal mastitis in the goat, was able to come to a more definite conclusion as to the nature of the accessory glands of the teat. These glands were present in 18 of the 29 goats examined, the extent of the glandular tissue varying from goat to goat. In non-lactating goats this tissue, like the mammary tissue proper, was in an involuted state and in animals inoculated with streptococci the abnormalities of the mammary tissue proper caused by the bacteria were present also in the accessory tissue. Pattison concluded from this that the accessory tissue is true secreting mammary tissue which does not differ from the mammary secreting tissue as a whole. Blackburn (1952), studying bovine mastitis, found that these accessory lobules, as well as occurring in the cistern walls, occured also in the walls of the ducts. They were termed *periductal lobules*. The present work was done to obtain further information on the distribution of periductal lobules.

Method

In order to make a reconstruction, serial sections were cut at 7.5μ from a block of involuted udder tissue. Alternate sections were mounted and therefore each represented 15μ of the tissue. White cardboard 0.8 mm. thick was used to make the model, which meant that the magnification required was $800/15 = 53.3$ times. This magnification was obtained by using a 2 in. objective with the draw tube of the microscope drawn out to its full length (21 cm.), and the drawing-board placed 14 cm. below the camera lucida mirror. The method of Gage (1907) was used in making the model, the main modification being that cardboard was used instead of blotting-paper.

The outlines of ducts were first drawn on paper and then traced on to cardboard. The shapes were cut from the cardboard by means of a sewing-machine using a short "stitch" and a bayonet-pointed needle. The shapes were stuck to those of the previous level by means of quick-drying glue (Durofix), the paper drawing being used as a template to indicate the correct positions. It was found that strengthening pins were not required. Plastic wood was used to smooth off the steps between the levels, and the model was finished with

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(Article by Blackburn, page 366)

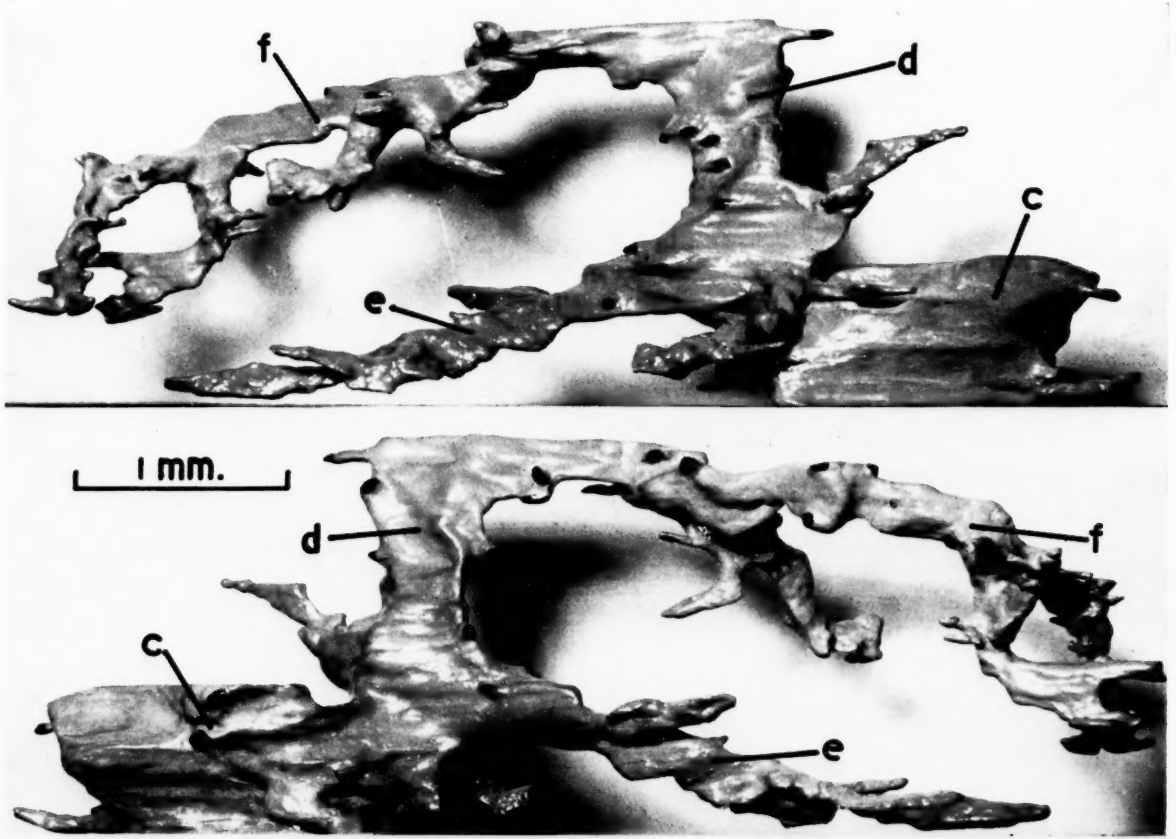

Fig. 1 Two views of a reconstruction of the finer ducts of the bovine mammary gland. The lobular ducts of periductal lobules are painted black.

carborundum wheels in a portable high-speed grinder and painted with cellulose paint. The lobular ducts draining periductal lobules were painted black.

Results

Views of opposite sides of the reconstruction are shown in Fig. 1 (*a*) and (*b*). For descriptive purposes the reconstruction may be said to consist of two "vertical" and two "horizontal" ducts. The larger vertical duct (*c*) seen on the right of Fig. 1 (*a*) was only partially reconstructed. In the tissue the transverse section of this duct measured 1.3 x 0.5 mm. The smaller vertical duct (*d*) which measured 0.5 x 0.1 mm. in the tissue, drained into the larger vertical duct and close to the junction there arose five ducts which drained from two to seven lobules. The lower of the two horizontal ducts (*e*) also arose from this region. Reconstruction of the lower horizontal duct was not complete. The smaller vertical duct (*d*) was reconstructed to the point where the block of tissue terminated. Before this point was reached, however, the upper horizontal duct (*f*) was given off and this was reconstructed in its entirety.

Periductal lobules.

Two of the lobular ducts which drained the periductal lobules can be seen on the border of the larger vertical duct (*c*) in Fig. 1 (*a*) and two others can be seen on the side of the duct shown in Fig. 1 (*b*). There are three at the origin of the lower horizontal duct (*e*) and nine arising from the smaller vertical duct (*d*). The lobular ducts are concentrated on the borders and on the surface of the latter duct and can be seen in Fig. 1 (*a*). It can be seen from Fig. 1 (*b*) that there were none on the opposite surface. In fact, on that side the larger vertical duct passed within 0.5 mm. of this surface and therefore there was not room for lobular development. In the wall of the upper longitudinal duct (*f*) there are eight lobular ducts which drained periductal lobules.

Terminal lobules.

The upper horizontal duct (*f*) showed the distribution of ducts to the terminal lobules. About halfway along, the duct gave off a side branch and then continued until it finally divided into two terminal branches. The side branch and one of the terminal branches drained irregularly-situated lobules, whereas the other terminal branch, seen on the extreme left of Fig. 1 (*a*), drained a series of paired lobules which were more regularly situated. This difference in distribution may have been developmental in that this branch was an original growing tip free to develop lobules, whereas the other two branches appeared later and developed lobules where space was available.

The relative importance of ducts as seen in sections of udder tissue.

An interesting point, which is perhaps worthy of mention, was noticed whilst making the reconstruction. The size of a duct in a section gave no indication of its relative importance. For example, longitudinal sections of the horizontal ducts (*e*) and (*f*) were larger in some instances than transverse sections of the vertical duct (*c*) which was the most important of the three.

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Discussion

As well as the so-called accessory lobules which occur in the walls of the teat cistern and gland cistern of the bovine mammary gland, periductal lobules occur along the walls of the duct system to the point where the terminal branches which drain the terminal lobules arise. In a fully-lactating udder most of the space which is not taken up by ducts, blood-vessels and lymphatics is occupied by lactating lobules. It is probable that the development of periductal lobules is necessary to achieve this. From celluloid casts of the duct system of the udder Blackburn (1952) found that after primary branching the ducts tend to radiate to the periphery of the quarter and to meet those of the other quarter on the same side of the udder. It is as if in development there is an attempt to gain as much tissue as possible before the intervening spaces are filled in by the growth of side shoots of varying size. Finally, in development there is only space left in the udder for the formation of periductal lobules. By means of reconstructions of individual lobules Weber, Kitchell and Sautter (1955) have studied the arrangement of intralobular ducts. They found that the method of branching was variable, but the duct entering the lobule usually divided into two or three branches. or in some instances five or six branches, and as a rule these branches extended for a considerable distance into the lobule. It is evident from their description that the arrangement of the intralobular ducts is similar to that of the larger ducts of the udder. These authors describe alveoli draining into terminal ducts and, in addition, what they have termed duct-alveolar combinations in which one or more alveoli are found interspersed in the course of an intralobular duct. It would seem that the duct-alveolar combinations arise from the outgrowth of one or more alveoli from the duct wall in a similar manner to the growth of a lobule from a duct wall, and for the same purpose, namely, the utilisation of available space.

The recognition of periductal lobules is of importance in the study of the histopathology of mastitis especially with reference to the spread of the lesions through the udder.

The occurrence of periductal lobules as seen in sections of udder tissue has given rise to the misconception that lobular ducts drain into dilatations or milk-collecting spaces in the ducts. Turner (1952) shows a diagram (Fig. 81, p. 130) of a so-called lobe in which four lobules drain into a collecting space. In fact the diagram is of four periductal lobules draining into a duct cut in transverse section. Weber, *et al.* (1955) report that, apart from duct-alveolar combinations, they found no milk-collecting spaces in the intralobular duct system similar to those described by Turner (1952).

The arrangement of the lobules in the bovine mammary gland has been likened to the leaves on a tree but it should be noted that the leaves, as well as being at the ends of the twigs, also arise from the sides of the branches and the trunk.

Summary

By means of a reconstruction of the finer ducts of the bovine mammary gland the distribution of periductal lobules has been studied. It has been

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established that the lobular duct of a periductal lobule drains directly into a larger duct in contrast to the lobular ducts of terminal lobules, several of which unite to form a larger duct. Periductal lobules were found to occur around all the ducts of the mammary gland. Such lobules occurring in the walls of the teat cistern and gland cistern have been known in the past as accessory glands.

Acknowledgment

I wish to express my thanks to Dr. J. D. Findlay for photographing the reconstruction.

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THE DEVELOPMENT OF RUMEN PAPILLÆ IN CATTLE FED ON DIFFERENT DIETS

By A. BROWNLEE, B.Sc., M.R.C.V.S.
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Barcroft, McAnally and Phillipson (1944 *a* and *b*) have shown that fatty acids are absorbed from the rumen of sheep and point out that rumen papillæ increase the absorptive area of the mucous membrane; they describe the stratified epithelium covering these papillæ, observing that this differs from the skin stratified epithelium as follows: (*i*) the layer of cornified cells is thin; (*ii*) the layer of granular cells, the *stratum granulosum,* although distinct in many places is by no means a continuous layer, (*iii*) no sebaceous glands are present, consequently there is no greasy protection to assist in preventing the passage of water through the epithelium.

The present work started when Dr. W. S. Gordon and Dr. J. H. Taylor made available post-mortem material from calves which had been the subject of an antibiotic experiment. Four groups of calves had been fed from birth for eight weeks and twelve weeks on a diet of milk only, in generous quantities, and for eight weeks and twelve weeks on a diet of milk, concentrates and hay ("ordinary diet"). The calves had been bedded with peat-moss and those in the two milk-only groups showed by their rumen contents that they ate some of this bedding. The calves on milk only showed no development of rumen papillæ, whereas those on the ordinary diet showed well-marked papillary development. The examination of these animals will be referred to as Observation 1.

Further work was carried out in order to find out what stimuli lead to the development of these papillæ.

Animals and Methods

Experiment 1.

Ayrshire and Ayrshire-cross cattle were used.

"Hay Group." Four maiden heifers about 2 years old and six heifers about 2 years 8 months old, five with calf at foot (1 to 2½ months old) and one which had aborted, were kept on a diet of hay and crushed oats plus grain-balancer compound cake. The period was four to five months in the case of the calves and four to six months in the older animals. The foods were not weighed; the approximate amounts given daily were:—

Maiden heifers. Eight pounds increasing to 12 lb. of concentrates and 16 lb. of hay.

Heifers with calves. Twelve pounds increasing to 18 lb. of concentrates, while with calves (15 lb. after calves were slaughtered) 16 lb. hay.

"Kale Group." Six maiden heifers about 2 years old and six heifers about 2 years 8 months old, each with calf at foot (1 to 2½ months old) were kept on a diet of kale and concentrates (similar to those fed to the "Hay Group") for 10 weeks, followed by a diet of mangolds and concentrates for a further 2½ to 5 months. The approximate amounts given daily were:—

Maiden heifers. Eight pounds increasing to 12 lb. of concentrates, plus 40 lb. kale or 64 lb. mangolds (cleaned and sliced).

Heifers and calves. Twelve pounds increasing to 18 lb. concentrates while with calves, 15 lb. afterwards, plus 40 lb. kale or 64 lb. mangolds.

In each group the calves were allowed to suckle their dam and share her food. All the animals were fed twice daily. The "Hay Group" was bedded with straw and the "Kale Group" with peat-moss. Wheat straw was used as bedding for two periods of about three weeks and seven weeks when no peat was available. The straw was mixed with the soiled bedding in an endeavour to discourage the animals from eating it; even so, they ate some.

Experiment 2.

Twenty-four bull calves, about 1 week old, were divided into four groups, bedded with peat-moss litter and fed twice daily in the case of the milk and milk substitute, with other foods *ad lib.*, for 12 weeks, as follows:—

Group 1. Milk diet. Six calves (five Ayrshire or Ayrshire-cross and one Friesian) on a diet of whole milk and milk substitute ("milk diet"), fed in increasing quantities.

Group 2. Milk and hay diet. Six calves (Ayrshire or Ayrshire-cross) on milk diet and allowed meadow hay.

Group 3. Milk and grass diet. Six calves (five Ayrshire or Ayrshire-cross and one Friesian) on milk diet and allowed fresh grass.

Group 4. Milk and concentrates diet. Six calves (three Ayrshire or Ayrshire-cross and three Friesian) on milk diet and allowed proprietary compound calf food.

At the end of the experimental period a number of the animals in Observation 1 and all those in Experiments 1 and 2 were examined on slaughter. The

following dissections were done in order to weigh the compartments of the stomach and to find the weight and length of the small and large intestine. The mesenteric attachments, exposed lymph glands and gross fat were removed, except in the case of the large intestine, where no attempt was made to remove visceral fat. The contents of the intestines were removed by stripping by hand and those of the stomach compartments by washing. The latter were pressed by hand in order to discharge easily removable water. The reticulum was separated from the rumen by cutting along the junction between the honeycomb mucous membrane of the reticulum and the papillary mucous membrane of the rumen. In Experiments 1 and 2 the rumen mucous membrane was separated from the muscular coats by manipulation. The animals in Experiment 1, which were older, had a quantity of fat deposited on the surface of the rumen and between the folds of the pillars. The remains of this fat were removed before the rumen muscle was weighed. It was observed that the weights of the mucous membrane plus muscle (plus fat in Experiment 1), did not amount to the weight of the whole rumen, the small loss presumably being due to evaporation.

For the purpose of this present communication, data relating to the rumen alone are shown (Tables 1 to 3). It is hoped to include the other data in a future publication.

TABLE 1

AVERAGE AND RANGE OF LIVE WEIGHTS AND RUMEN WEIGHTS OF ANIMALS EXAMINED IN OBSERVATION 1

AGE DIET NUMBER OF ANIMALS EXAMINED :	8 weeks old "Ordinary" rations 4	8 weeks old Milk 6	12 weeks old "Ordinary" rations 20	12 weeks old Milk 9
LIVE WEIGHT				
Average	135.2 lbs.	185.3 lbs.	180.6 lbs.	240.1 lbs.
Range	126 - 154 lbs.	165 - 204 lbs.	134 - 222 lbs.	180 - 347 lbs.
RUMEN				
Average	608 grms.	389.6 grms.	1149.8 grms.	584.3 grms.
Range	522 - 780 grms.	305 - 495 grms.	855 - 1675 grms.	412 - 897 grms.

Results

Fig. 1 shows a typical specimen of part of the floor of the anterior, dorsal, blind sac of the rumen and of the mucous membrane of the reticulum of an animal from the 12 weeks ordinary ration group in Observation 1, where the rumen papillæ are well developed, and the corresponding rumen tissue from an animal in the milk-fed group, where the papillæ are undeveloped. Typical specimens of corresponding mucous membranes from each of the four groups in Experiment 2 are shown in Fig. 2; the milk-only-fed animals show no papillary development, whereas the hay group show papillæ measuring up to about 0.5 cm. long, the grass group up to about 0.7 cm. long, and the concentrates group up to about 0.8 cm. long. The papillæ in the hay and the grass groups are more slender than are those in the concentrates group. In the concentrates group the mucous membrane tore readily when being stripped off the muscular coats, whereas in the hay and the grass groups it was more tensile. In the con-

TABLE 2

EXPERIMENT 1.

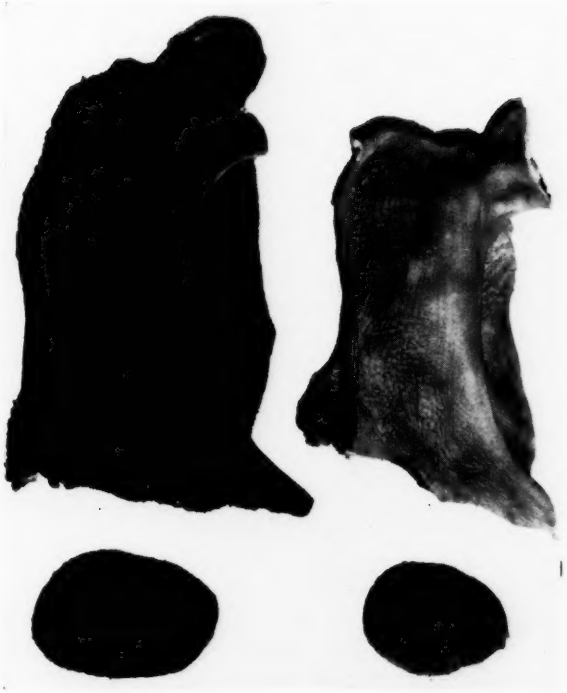
Group	Maiden Heifers		One calf Heifers		Calves	
	"Hay"	"Kale"	"Hay"	"Kale"	"Hay"	"Kale"
Number in group	4	6	6	6	5	6
Live weight: average	8.891 cwts.	8.166 cwts.	9.135 cwts.	8.708 cwts.	4.35 cwts.	3.875 cwts.
" " range	8.063 - 10.25 cwts.	7.5 - 9.0 cwts.	8.0 - 11.25 cwts.	8.0 - 9.5 cwts.	4.0 - 4.75 cwts.	2.75 - 5.0 cwts.
Rumen weight: average	7107 gms.	6961 gms.	7836 gms.	7859 gms.	2737 gms.	2780 gms.
" " range	6810 - 7604 gms.	5902 - 7718 gms.	6696 - 10669 gms.	7320 - 9023 gms.	2185 - 3178 gms.	1802 - 3972 gms.
Rumen mucous membrane : average	2838 gms.	3270 gms.	3169 gms.	3693 gms.	1022 gms.	1233 gms.
Rumen muscle: average	3589 gms.	3041 gms.	4015 gms.	3386 gms.	1257 gms.	1123 gms.
Rumen mucous membrane expressed as a percentage of live weight:						
Frequency Table .36 - .49	1				4	1
.50 - .57			1		1	2
.58 - .65			1			1
.66 - .73	2	2	2	1		1
.74 - .81	1	2	2	3		1
.82 - 1.03		2		2		
Average percentage :	.64	.77	.67	.84	.46	.61
Rumen mucous membrane expressed as a percentage of rumen mucous membrane plus rumen muscle:-						
Frequency Table 38 - 41	1		1		1	
42 - 45	2		3		2	1
46 - 49	1		2	1	2	
50 - 53		6		3		1
54 - 57				2		4
Average percentage :	44	51	44	52	45	52

TABLE 3

EXPERIMENT 2.

Group :	1	2	3	4
Diet :	Milk	Milk + Hay	Milk + Grass	Milk + Concentrates
Number in Group:	6	6	6	6
Live weight : average	148.8 lbs.	154 lbs.	175.6 lbs.	191 lbs.
" " range	108 - 179 lbs.	94 - 186 lbs.	163 - 187 lbs.	156 - 210 lbs.
Rumen weight : average	640 gms.	1056 gms.	1448 gms.	1454 gms.
" " range	496 - 799 gms.	640 - 1287 gms.	1238 - 1645 gms.	1021 - 1643 gms.
Rumen mucous membrane : average	148.9 gms.	347 gms.	561 gms.	720 gms.
Rumen muscle : average	481 gms.	701 gms.	846 gms.	707 gms.
Rumen mucous membrane expressed as a percentage of live weight :				
Frequency Table .187 - .261	6			
.472 - .543		6		
.576 - .675			2	1
.676 - .775			3	
.776 - .875			1	4
.876 - 1.027				1
Average percentage	.224	.496	.702	.826
Rumen mucous membrane expressed as a percentage of rumen mucous membrane plus rumen muscle:				
Frequency Table 19 - 24	3			
25 - 30	3			
31 - 36		6		
37 - 42			5	
43 - 48			1	3
49 - 54				3
Average percentage	24.02	33.25	39.79	50.18

PLATE I



No. 50 — Age 12 weeks. Ordinary diet. Live wt., 170 lb.; rumen wt., 1162g.; reticulum wt., 150g.

No. 499 — age 12 weeks. Milk diet. Live wt., 267 lb.; rumen wt., 740g.; reticulum wt., 90g.

Fig. 1

Portion of floor of anterior dorsal sac of rumen and mucous membrane of reticulum of two calves in Observation 1.

(*Article by Brownlee, page* 369)

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PLATE II

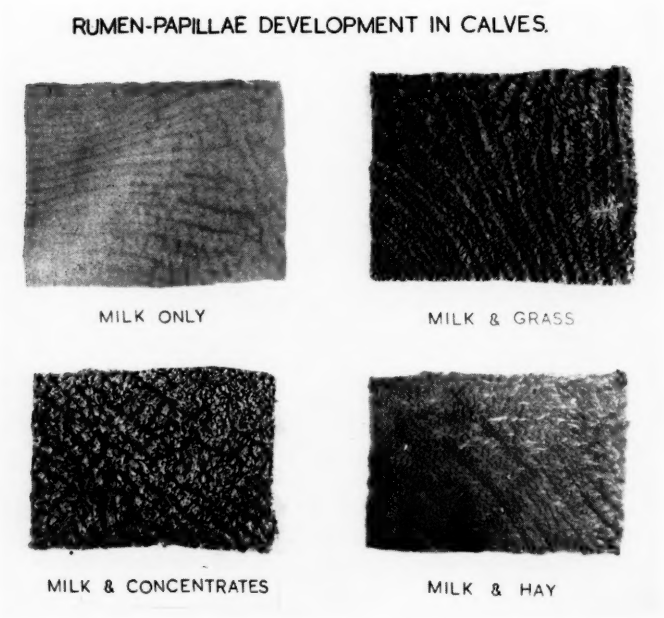

Fig. 2
Portions of mucous membrane of rumen from calves in Experiment 2.

(*Article by Brownlee, page* 369)

centrates group the mucous membrane covering the posterior pillar showed areas of superficial erosion, whereas such lesions were absent from the other three groups of Experiment 2. These experimental studies do not show whether such lesions would be likely to occur in animals fed on concentrates according to accepted farming practice, when the concentrates would be fed along with other solid food.

The weights of the rumen from six Ayrshire or Ayrshire-cross calves whose ages ranged from 2 to 22 days were found to average 135.5 gm., with a range of 120 to 162 gm; a comparison of these weights with the rumen weights of milk-fed Ayrshire or Ayrshire-cross calves in Observation 1 (see Table 1) shows an increase in weight in the latter over their probable weight at birth of up to as much as over six times (i.e., 135.5 gm. against 897 gm., the heaviest milk-fed rumen in animals examined from Observation 1); yet no development of papillæ had occurred. Further evidence that the development of papillæ is not a function of development of the rumen as a whole is to be found by comparing the average of the weights of rumen mucous membrane and rumen muscle in Groups 2, 3 and 4 of Experiment 2 (see Table 3). The average mucous membrane weight in Group 4 (720.1 gm.) is considerably heavier than in the other two groups (347.1 gm. and 561.3 gm.), yet the average muscle weight in Group 4 (707.1 gm.) is actually less than in Group 3 (845.6 gm.), and only slightly greater than in Group 2 (700.75 gm.).

It is necessary to point out here that all the examinations carried out indicated that when, to visual inspection, there appears to be a greater development in the size of the papillæ then the weight of the whole mucous membrane, after being stripped off the muscular coats, will be found to be heavy in relation to the weight of the rumen as a whole. It is being assumed that the mucous membrane weight is an index of papillary development, and that the only accurate way of expressing papillary development would be in terms of weight, as papillæ vary considerably in size in the different areas and they may also vary within any given area.

In Experiment 1 the average of the mucous membrane weights of the "Hay Groups," expressed as a percentage of the average total live weight, is less in the calves and in the two groups of older animals than the corresponding figures for the "Kale Group" (see Table 2). This frequency table also shows that in all three ages of animals a greater number of mucous membrane percentage figures fall in the higher percentage columns in the "Kale Group" than in the "Hay Group."

Similar results are obtained when one compares in these two groups the mucous membrane weights expressed as a percentage of the aggregate of rumen mucous membrane plus muscle (see Table 2). These results indicate that roughage in the form of hay was not an important factor in developing rumen papillæ, as, indeed, the lower-roughage kale and/or mangolds acted as the more powerful stimulus. Eight of the older animals in the "Kale Group" had been bedded with peat-moss continuously for periods varying from three to seven weeks immediately prior to slaughter, and in every one of these the rela-

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tively better developed mucous membrane was still found, so that it is improbable that the access to some straw bedding earlier in the experiment was an important factor in mucous membrane development.

It became clear during the course of this work that for experimental purposes it is unsatisfactory to feed more than one rumen-entering food to any given animal, as the effect of one food may obscure that of another; hence Experiment 2.

The results of Experiment 2 (Table 3) show that the average weight of the rumen mucous membrane expressed as a percentage of the average body weight increases from 0.224 per cent in Group 1 to 0.496 per cent in Group 2, to 0.702 per cent in Group 3 and 0.826 per cent in Group 4. The character of the mucous membrane did not appear to be influenced by breed difference. Frequency tables of these results (Table 3) show that a greater number of mucous membrane percentage figures fall in the higher percentage columns in the hay group compared with the milk-only group, in the grass group compared with the hay group, and in the concentrates group compared with the grass group.

Discussion

That morphological changes can occur as an adaptation to environmental stimuli is well known, obvious examples being the changes in the growth of hair in response to climatic variation and changes in the thyroid in response to the level of available iodine. So also, the present work on rumen papillæ indicates that the extent of their development is not predetermined, but varies with the nature of the diet. Early in this work it was thought that roughage possibly was a factor in developing these papillæ, but both Experiments 1 and 2 point to the reverse being the case, namely, that the more fibrous the diet, as peat-moss and hay, the less the growth of the papillæ, while the less fibrous foods, such as kale or mangolds, grass and concentrates, gave rise to a greater development. Thus it would appear that what determines the degree of development is more likely to be the energy value of the food or the rapidity with which it is broken down into absorbable fractions.

These observations deal with the influence of food on the development of the rumen mucosa. It is interesting to consider these findings in relation to the animals' behaviour. The milk-only-fed calves showed that they ate a quantity of peat-moss, a substance not eaten by cattle except when suitable food is not available. This behaviour occurs even in calves receiving a generous quantity of milk, and indicates a drive to eat solid food. Under normal circumstances the consummation of this drive would be the filling of the rumen with papillæ-stimulating food. In a previous paper (Brownlee, 1954) I pointed out another drive in calves, namely the play drive the consummation of which results in functioning, with presumably accompanying functional hyperæmia, of certain neuro-muscular groups which in the absence of play would not be fully exercised in the young animal, and consequently not fully developed. Thus, as in the case of these muscle groups, so with rumen papillæ, it would appear that behavioural activity is required in order to achieve their full development.

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Summary

(1) Cattle on a diet of kale plus concentrates, followed by a diet of mangolds plus concentrates, showed greater development of rumen papillæ than did similar animals on a diet of hay plus concentrates.

(2) Newborn and week-old calves kept on a milk diet and bedded with peat-moss did not show development of rumen papillæ up to at least 13 weeks of age, whereas week-old calves on a diet of milk plus hay, milk plus grass, and milk plus concentrates, for 12 weeks in each case, showed development in increasing degree. Thus development of these papillæ is not predetermined, but is dependent on the nature of the food.

(3) In calves kept on a milk diet and bedded with peat-moss the rumen as a whole can increase in weight as much as some six times its probable weight at birth without corresponding papillary development; thus the rumen muscle development is not *pari passu* with papillary development.

(4) Roughage as such is not a factor in developing rumen papillæ. The indications are that development is determined by the energy value of the foods or the rapidity with which they are broken down into absorbable fractions.

(5) The rumen mucous membrane in calves fed concentrates without the addition of other suitable solid food was found to be easily torn, and also areas of superficial erosion of the mucous membrane covering the posterior pillar were observed.

(6) The role played by the animal's behaviour in determining organ development is discussed briefly.

Acknowledgments

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VETERINARY PATHOLOGY: Some Lessons from the Past

By J. F. SMITHCORS,* D.V.M., Ph.D.

THIS paper is the outgrowth of two seminars on the history of veterinary pathology presented upon the invitation of the Department of Pathology of the College of Veterinary Medicine at Michigan State University. At the outset I should state that I am not a pathologist, but that I do have an interest in the subject from the standpoint of the broad aspects of veterinary education, with a particular penchant for its historical development. While the historian would hope there might be an occasional individual with an interest in the development of his specialty for its own sake, it is, perhaps, logical that the greatest good will be realised if the historian can demonstrate some utility of history to his colleagues. I have, therefore, pursued the premise that aside from any intrinsic interest the pathologist might derive from a study of the past, there are, for those concerned with veterinary education in particular, some concrete lessons to be learned. And inasmuch as the veterinary practitioner is, or should be, to a greater extent than the general practitioner of medicine, a "comparative pathologist" in the fullest sense, there are lessons for him also.

In reviewing some general pathology, I was intrigued by a passage from the preface of Boyd's *Textbook of Pathology* (1943). This passage, which I had underlined as a student, reads:—

". . . the pathologist may have to content himself with applying a great variety of names to an equally great number of states, the relationship and meaning of which he really does not understand. The student must learn to recognise that ignorance, however aptly veiled in an attractive terminology, still remains ignorance."

How true this may be to-day—and this was not really so long ago—I shall leave for the pathologist to decide. This historian who is not also a specialist in the field he is treating treads upon dangerous ground when he attempts to assess the developments which have occurred within the lifetime of the elder statesmen of that specialty. The present study, therefore, is limited largely to developments prior to this century.

The humoral theory of pathology, as elaborated by Galen in the first century, did much to retard the practice of medicine until Morgagni's epochal work in 1761 provided the foundation for a pathology based upon changes in organs. The tissue pathology of Bichat (1832) and Henle (1846) paved the way for the cellular pathology of Virchow (1858), and the development of bacteriology later in the century brought about an experimental approach to pathology. The early microscopic studies were done with fresh, unstained tissue sections, and while the development of dyes used in conjunction with fixed tissues made possible great strides in increasing the intimate knowledge of cell structure, more recent emphasis upon the fluid medium of the cell has revitalised the study of fresh tissues. In a sense, then, we are witnessing the completion of a two-

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thousand-year cycle, not, certainly, a return to a conjectural humoral pathology based upon "melancholy," but, instead, a chemical pathology based upon molecules. As a matter of passing interest, a writer in the *Veterinarian* for 1861 called for a return to humoral pathology because of the interest in the blood as the seat of diseases. Had Galen been permitted the luxury of human post-mortem examination, it would seem inevitable that the history of medicine over the period of some seventeen centuries following his time would have been vastly different. Considering the circumstances, Galen's humoral theory was, perhaps, the best that could be devised, and certainly was better than no theory at all. The dogmatic acceptance of his ideas continued to exert a powerful retarding effect upon medicine long after post-morten examination came to be practised with some regularity. But, as too often is done, Galen, himself, cannot be blamed for the failure of later men to make better use of material at their disposal, but which was denied him.

This brings us to our first major tenet, namely, that the regular practice of post-mortem examination is essential to the development and continuance of a sound system of medicine, to say nothing of pathology itself. This, of course, is in the nature of a cliche to the professional pathologist. But is every student in his greener days sufficiently imbued with this doctrine? Does the student invariably perform, or at least see the results of the examination of cases he has attended which have terminated unfavourably? Or are there times when he may secretly be glad that some necessity, more apparent than real in most cases, excuses him from the "messy business," and the unprotesting and unopened cadaver becomes the prized booty of the knacker? And if he should have been permitted some laxity as a student, will the magic mantle of the diploma transform him into a true "comparative pathologist" who will be faithful to himself and to his clients?

Veterinarians of the remote past apparently recognised their obligations in these matters, for Aristotle speaks of the lungs being the seat of a condition which we can recognise as pleuropneumonia of cattle. And the Greek veterinarians of the Byzantine era produced a system of veterinary medicine superior to that of human practice, perhaps largely because they, too, learned their pathology first-hand, instead of being content with armchair compilations. Countless generations of later veterinary practitioners, hardly worthy of the name, became more interested in cures than in causes, and the veterinarian became a non-entity. Not until the 18th century do we find a mounting dissatisfaction with the degraded status of veterinary medicine—at which time the gulf between human and veterinary medical practice had widened to a veritable chasm. And then the first voices to be heard with any consistency were those of men who had some medical training. Of this group, J. Bartlet, a surgeon who had turned to veterinary writing, if not practice, for all of his faults, offers a commendable plea for the regeneration of veterinary pathology. In his *Gentleman's Farriery* (1753), he states:—

"It cannot indeed be expected that our farriers should make any real progress in their profession till they apply themselves early to the study of

anatomy, and to the dissections of morbid bodies, and lame horses: by this method, and only this can do it effectually, a new scene of knowledge will be unfolded to them . . . But now, through ignorance of the real seat of the disorder, how frequently do they rowel, cauterise, and apply medicines externally, to parts free from any ailment or grievance whatever, while they neglect the real source of the disorder? . . .

"But till this study is more patronised among us. we must not wonder at the slow advances it makes; for the practice and character of a horse physician, or surgeon, at present stands so low in the popular opinion . . . that not even a country surgeon, who is almost starving under a scarcity of human patients, will deign to attempt a fortune by applying his medicinal and chirurgical talents to the service of this necessary servant of mankind."

Other medical men urged the study of comparative pathology as a basis for explaining the diseases of both animals and man, but veterinary practice was for some time yet too firmly entrenched in the hands of men like William Taplin. In spite of his medical training, Taplin became the prototype of the worst sort of horse-doctor who proudly boasted he had "never lost a case." Of Taplin, who described the gall-bladder of the horse and its diseases through some fifteen editions of his *Gentleman's Stable Directory* (1788), General Smith (1927) says:

". . . his practice was so successful that he never appears to have been able to make a post-mortem examination in order to correct his anatomy . . . He bequeathed nothing to posterity but a well-hated name and a detestable personality."

Despite an apparently large practice, Taplin boasted that no horse had ever left his establishment dead—which makes one wonder if he might have had a large cellar, preferably with a tightly-fitting trapdoor.

So long as men of the stripe of Taplin flooded the market with worthless works bearing opprobrious titles, little progress might be expected to be recorded, for the status of the profession is mirrored in the works produced by and for the practitioner. A high standard of practice is dependent first of all upon the calibre of the men who are the teachers of any particular generation of students, and who, frequently enough, are the authors of the works which will guide their graduates in later years. As a second major tenet, then, we might recognise the fact that the present recognition of veterinary pathology as a discipline in its own right depends to a large extent upon those publications which unhesitatingly proclaim themselves as the standard-bearers of the subject and, of course, render more than lip-service to the cause. In passing, it might be noted that no distinction between "veterinary" and "comparative" is intended. nor should it be inferred with relation to animal pathology; it was a little surprising, however, to find that the Michigan State University Library places the *Journal of Comparative Neurology* with the veterinary publications.

The first British veterinary text which claimed to be a pathology of sorts was William Ryding's *Veterinary Pathology* (1801), which General Smith describes as a worthless rehash of notes taken at the London Veterinary College. Much

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the same might be said of the first American reprinting of a British work in this area, *A Compendium of Anatomy, Physiology and Pathology of the Horse* (1806), by B. W. Burke. On the other hand, the first British text on human pathology, Matthew Baillie's *Morbid Anatomy of Some of the Most Important Parts of the Human Body* (1793), is described by Mettler (1947) as a "disappointing little book." She further states that systematic pathology was notoriously deficient in the original medical texts of the period. Had James Clark completed his *First Outlines of Veterinary Physiology and Pathology* (Vol. I, 1806), it undoubtedly would have been a substantial contribution, but the first volume deals mostly in generalities, primarily from human sources. The first special veterinary pathologies appear to be Delabere Blaine's *Canine Pathology* (1817) and William Percivall's monumental *Hippopathology* (1834-1852), the latter being a standard work both in Britain and America for many years. Original American works on veterinary pathology are conspicuous by their absence until the early part of the present century, but this may be attributed to the slow start veterinary education received in this country. In this country, Runnell's *Animal Pathology* (1938, 5th ed. 1954) is the standard text. In the field of veterinary periodical literature, William Youatt's frequent contributions on the subject in the *Veterinarian,* from 1828 on, did much to advance the concept of animal pathology.

While veterinary pathology as a discipline in its own right depends upon contributions to the subject being identified as such, other writers on the more general subject of animal disease made substantial additions to pathology. Although the first edition of his *Outlines of the Veterinary Art* (1802) had serious shortcomings in fact, Blaine may be credited with being the first British veterinary writer to present a reasonably acceptable system of veterinary medicine, including pathology. His later editions live up to the promise offered earlier. Concerning the much-needed reform of veterinary practice, he says that farriers:—

"... always began where they should have ended: they gave rules for the treatment of disease, but they never taught what the disease was by explaining the structure, function and economy of the animal body in health—a superstructure without a base ...

"All the writers that have appeared in veterinary medicine to this time have considered disease as arising from an affection of the fluids of the body ... the lack of improvement in veterinary medicine is almost wholly due to this obstinate adherance to humoral pathology, by which the treatment of internal and acute diseases continues inert and unsuccessful."

As a corrective measure, he urges that "a general acquaintance with human pathology will greatly assist the veterinarian."

While General Smith (1930) is justly harsh on certain of Blaine's unprofessional characteristics evident in his earlier works, he states, concerning *Canine Pathology*: "Had he never written anything else, it would have placed him among the elect," and that the 1832 edition of his Outlines is "a really good work beautifully written and expressed." Smith's evaluation of Blaine's work

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accords him a measure of the recognition of which he was deprived, for a number of reasons, during his lifetime; for:—

"Fortunately Blaine had been trained in a school of experimental inquiry, and it was the adoption of this method which enabled him to progress on sound lines, for he employed it in his practice, and advises others to do the same, but not to talk about it. He stresses the necessity of post-mortem examinations, of which he made some hundreds in rabies alone . . . Can it be doubted that had our school carried out its work on the lines of experimental research and post-mortem exactitude, we should have been spared the long years of guessing and worthless conjecture which have done so much to retard the growth of the profession?"

That the London school demonstrated some obvious deficiencies in its early years is sufficiently well documented by General Smith, and by Youatt and others. in the *Veterinarian,* to require little more than recognition of the facts here. The three-year programme dealing with all classes of animals, instituted by Sainbel, who urged the necessity of a high calibre student, was cut to little more than a three-month's study of the horse by his surgeon-successor, Edward Coleman, who favoured the student of lesser capabilities. The curtailed programme of study he defended upon his claim that the diseases of the horse were few and simple; he openly despised comparative anatomy, saying that it led to "comparative physiology and comparative pathology." On the other hand, this situation was not peculiar to the London school, nor to one side of the Atlantic. Long after both reason and precedent would have dictated a broader scope of veterinary education in America, veterinary colleges remained horse-centered, and did little to strive for higher standards until these were forced upon them by outside agencies.

The first to wage a relentless fight for higher standards of veterinary education in Britain was William Youatt, through the medium of the *Veterinarian,* and, by supplying through his private school, some of the teaching that was lacking in the London Veterinary College. Youatt's argument for increasing the period of study was based primarily upon the need for instruction in comparative pathology. Although he approved of the general plan instituted under Sainbel, he nevertheless was critical of Sainbel's appointment, saying that Sainbel knew nothing of comparative pathology. But this was written some forty years after Sainbel's death, and it is doubtful that anyone, other than, perhaps, John Hunter, in Britain had an adequate grasp of comparative pathology in the 1790's. Despite the urgings of Youatt and others, the *status quo* was maintained until after Coleman's death in 1839, the first Professor of Cattle Pathology being appointed in 1842. Needless to say, this appointment came too late to save Great Britain from the devastation of the series of cattle plagues which had already begun.

The failure to develop the fundamental sciences as a strong part of veterinary education plagued the veterinary profession in both Great Britain and the United States for the first half-century or more after the establishment of

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formal veterinary education in either country. To a greater extent than any other single factor, this resulted not only in a low level of proficiency on the part of the practitioner, but it especially hindered his capacity for continued self-development after graduation. This is especially true of pathology; without discounting the need for sound training in anatomy and physiology, and the other sciences, the very nature of general practice is such that pathology is the heart of the training of the practitioner. That failure to graduate veterinarians who are practical pathologists creates a vicious cycle is evident from the commentary of W. A. Cherry, a medically educated veterinary practitioner, in the *Veterinarian* (1846). He states:—

"It is the general practice amongst medical men—and by consequence the public generally—to place the veterinary practitioner at a low ebb as to knowledge, that should any member of the latter art enter the lists as a physiologist or pathologist, he is set down as being most presumptuous, and whatever may be the truth or value of his observations, they are scouted as totally unworthy of attention."

He points out that the veterinarian has an immense advantage in being able to conduct post-mortem examinations immediately after death of the animal, and in all of his cases which so terminate, whereas in human practice, post-mortem decomposition is often confused with pathological changes, and all too few cases are examined. But he says he has met with:—

". . . so much envious malice and obstruction in the pursuit of veterinary physiology and pathology from human medical practitioners . . . (who) claim of being the whole and sole possessors of comparative medical lore; not only do they act upon this assumption, by never alluding to the researches of any veterinarian, but, still farther to mark the utter contempt in which they hold us, we are not considered fit, as *veterinary surgeons,* to mix with them."

Not all medical men, of course, shared this view, and by the mid-century some progress was being made in attacking the problem at its roots. But under the circumstances, progress was likely to be slow; just how slow it was in the next quarter-century may be adduced from the statement of George Fleming (1875) that:—

"It is curious to find that, while chairs are endowed at our universities for teaching the most abstruse and unprofitable subjects, speaking comparatively, veterinary science and the science of comparative pathology, which are capable of conferring the greatest possible advantages, in a humanitarian, scientific and utilitarian point of view, should be so strangely neglected."

His founding of the *Veterinary Journal* in 1875 might be thought of as a means of coping with this situation.

While pathology of a sort, usually in conjunction with surgery or therapeutics, was taught from the inception of the veterinary schools, the establishment of independent chairs of pathology was a late development. In the human field, Sir Astley Cooper had instituted post-mortem examination as a regular hospital teaching procedure in the late 18th century, and the first university chair of

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pathology was that occupied by Lobstein at Strasbourg in the 1830's. But this development by no means became universal for some time. Samuel Gross, the first eminent pathologist in America, complained in 1856 that in the 55 medical colleges in this country, there was practically no one teaching pathology. James Simonds was appointed the first Professor of Cattle Pathology at the London Veterinary College in 1842, but, according to Smith, "he was not a good pathologist." At Edinburgh, John Barlow was made Professor of Anatomy, Physiology and Pathology in 1844; under him, some major developments in the field of pathology might have been expected, but he died at a relatively early age. Professor Gairder, a leading pathologist at the University of Edinburgh, is quoted by Smith as saying that he often consulted Barlow and always with advantage. Other friends at the University expressed regrets that Barlow had chosen so "unworthy" a field for his endeavours. General Smith (1933) says of Barlow:—

"He was not only a skilled anatomist, but also a pathologist . . . His teaching had the special virtue of training men to reason and think, for he did not merely describe symptoms, but endeavoured to trace them back to the morbid conditions from which they arose."

While the French schools did not provide for special instruction in pathology at their founding, four chairs among the nine recommended in 1849 dealt with the subject. These were the chairs of : Pathological Anatomy, Sanitary Police, and Legal Medicine; Medical Pathology and Therapeutics; Surgical Pathology and Shoeing; and Clinics and Special Pathology, the latter presumably being post-mortem pathology. At the London school, G. W. Varnell, the distinguished Professor of Anatomy, in the 1850's pressed, unsuccessfully, for systematic instruction in pathology; this was not adequately realised until a Department of Comparative Pathology and Research was established in the 1890's. Varnell kept exhaustive post-mortem records, and urged others to do likewise, lest it be said that "we of the present generation are neglecting the opportunities of doing something for the benefit of those who may follow us."

In America, the abortive Philadelphia Veterinary College in the 1850's provided for a professor of Pathology and Surgery, and in the 1870's the New York College of Veterinary Surgeons had a Professor of Surgery and Surgical Pathology. Of eleven major schools in 1895, four had a Professor of Pathology, and three a Professor of Pathology and Bacteriology or Histology, all having the M.D. degree, rather than being veterinarians. One school had no chair which included pathology. Six, including some of the above, had veterinarians in chairs ranging from "Cattle Pathology and Obstetrics" to that of "Theory and Practice, Histology, Pathology, Helminthology, Bacteriology and Sanitary Science." Upon examining the status of the veterinary profession at this time, and following it through the past half-century, it would appear to be more than mere coincidence that the increasingly high standard achieved should have been accompanied by a similar development of veterinary pathology as an independent discipline. This does not mean that a professor with two, or four, or six titles cannot make a substantial contribution in one or more of his fields, but this is

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necessarily an age of specialisation, and in veterinary pathology itself, specialists are increasingly appearing. Much of the work in the future, in fact, will be done by teams of specialists; certainly, the pathologist is to an increasing degree dependent upon the biochemist and other scientists.

The contributions any one man can make in any field depend to some extent upon others having paved the way for him. But few would take issue with the statement that, in the English-speaking world at least, much of the improvement in veterinary medicine in the several decades after 1880 can be attributed to the work of Sir John M'Fadyean. Recognising in 1882 that, "we are yet far from occupying that position in the public regard that it is our ambition to attain," he stated (1937):—

"Our title to a higher place must rest on an increasing knowledge of the diseases affecting domestic animals, and on an increasing power to cure or prevent those diseases. Will it be believed that there is hardly an important disease peculiar to the domestic animals the pathology of which is fully and accurately known?

"That but little of this work has been done is because the individual members of our profession have not had that scientific training which is necessary to fit them for the task; and progress will only be made possible by the proper education of the veterinary student . . . whatever progress we have made, and are still making as a profession, may be traced to the development of veterinary education."

With courage in his convictions, but no suitable vehicle at his disposal, M'Fadyean instituted a course in pathology at the Dick school, on his own initiative and without remuneration. In 1888 the fruits of his energies were made available to an ever-widening segment of the veterinary profession, both at home and abroad, by the founding of the *Journal of Comparative Pathology and Therapeutics*. His choice of a title that called attention to the subject dearest to him might be looked upon as a carefully considered means of making up those deficiencies into which he perhaps felt that his own teaching was making but relatively slow inroads. In the journal for 1888, he stated:—

"What may be called veterinary pathology is at the present time in a very crude state as compared with the pathology of man . . . We often think with a great regret of the immense amount of valuable pathological material that in everyday practice is allowed to pass unutilised, in the sense that it is imperfectly investigated and not at all reported . . .

"It has been the bane of veterinary science . . . that everyone essaying to treat of a particular disease, however incomplete and imperfect his knowledge may be of the morbid anatomy of that disease, must formulate a theory which shall account for the symptoms. One nowhere finds a writer who has the courage to say: 'The pathology of this affection is quite unknown' . . . The recent graduate is too often launched into practice in the firm belief that there is little left for him to learn or discover . . . His faculties of observation and inquiry are thereby lulled to sleep, and great opportunities are therefore allowed to slip . . .

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To set forth the extent of our ignorance regarding any disease . . . is to perform a real service to pathology."

That M'Fadyean followed his own advice may be appreciated from the fact that in the twenty years from his appointment to the London school, he personally examined post-mortem every horse that died at the college. His teaching of pathology there developed to the point where in 1921, "a higher standard had been reached in this subject than at the medical schools." But medical critics of the veterinary profession claimed that comparative pathology was being neglected, and suggested a closer union of medical and veterinary education. M'Fadyean (1923) pointed out, with respect to earlier times:—

"It would be much nearer the truth to say that veterinary pathology was retarded because it was too much influenced by the current beliefs regarding human diseases . . . The greatest progress regarding animal disease has been made in the last fifty years, during which period veterinary pathology has, so to speak, commenced to stand on its own legs. The notion that it would be possible to create a class of men with an intimate knowledge of both human and veterinary pathology is a fantastic one, and it can hardly have occurred to anyone who has made himself a master of either. In both professions it is specialisation that has done most to increase knowledge."

Had these medical critics been as astute students of history as M'Fadyean was, they might have recalled the words of John Hunter, who, M'Fadyean (1923) says, "deserves to be called the father of comparative pathology," and whom he quotes as saying:—

"The arts of surgery and of human medicine alone can render very little service in cases of veterinary practice; while on the other hand, they may be, and frequently are, the source of error. . . . It requires the sacrifice of as many years to become a skilful physician; the acquisition of the science and the practice of each is a task sufficient to engage one man's life."

The pathologist of to-day can, of course, take a great deal of pride in the progress his profession has made and is making. Few of those whose bread and butter is earned by teaching the subject, and certainly none who engage in research, would so much as harbour the opinion that nothing is left to be discovered, or even that the end is, or ever will be, in sight. The thoughtful pathologist looks for remote, as well as immediate causes, whether he is conducting post-mortem examination, or investigating biochemical phenomena. But in looking for to-morrow's results, he may occasionally forget that those of yesterday may have some bearing on the problem he is trying to solve. Just as there is, in most cases, no basis for believing it unnecessary to look beyond the past two, or five, or ten years for data bearing upon a particular problem, so it is unrealistic to presume that still older records may not have a bearing, at least upon the broader aspects of the situation. Certainly the writings of John M'Fadyean, and John Hunter before him, on veterinary education and the scope of veterinary pathology are as valid to-day as they were when they were written. Had we accepted at face value Sir John's statement that, "progress

will only be made possible by the proper education of the veterinary student," there would be none graduated to-day with "the firm belief that there is little left for him to learn or discover." The situation in this respect is better to-day than it perhaps has ever been, but it would be worth while to pause to reflect upon whether we might be graduating some who are not sure of what might be left to discover, or conversely, what constitutes discovery. A fuller acquaintance with past history might serve to temper "new discoveries" made upon the basis of a single case report.

A timely warning is sounded by the editors of THE BRITISH VETERINARY JOURNAL (1951), who state: "There was a time in the history of veterinary science when much stress was laid on morbid anatomy and histology . . . The tendency seems to have been to devote less and less time and study to this aspect of disease, and to concentrate more upon cause than effect . . . We look forward to the time when there will be no cause to criticise the position of true pathologists in veterinary medicine . . ."

A century ago, the American veterinary practitioner, George Dadd, in his *Modern Horse Doctor* (1854), a work which was more professional than its title would indicate, said:—

"Some of our readers may think that there is no practical benefit derived from allusions to the unscientific customs of past days. But when we come to consider that the veterinary art has, in this country, progressed in a circle; that our domestic animals are still the subjects of misguided notions . . . this is our apology for alluding to the past, so that we may improve in the future.

We live in an age when the little we know of the veterinary art is a mere item of what we should know; for such knowledge can be made an instrument in the hands of philanthropic men for the benefit of all classes of creation."

Veterinary medicine in America was in a very crude state when Dadd wrote this; but while we need not be over self-effacing concerning the progress we have made, we cannot avoid realising that with regard to some problems, his statement has some application even to-day.

I do not suggest that every veterinarian should be an avid student of veterinary history, although a nodding acquaintance with the past cannot help but be of some service. The veterinary educator, however, should have more than a passing knowledge of the past of his profession in general, and his special subject in particular. A desirable graduate programme in veterinary pathology, therefore, would include course work, or, where this is not available, a planned programme of readings in the history of veterinary medicine, and of both human and veterinary pathology, and fuller attention to the broad background of the student's particular problem. One approach to realising this would be to have a doctoral candidate write a master's dissertation on the background and implications of his problem instead of confining himself to a review of the literature, in which primary, if not sole attention is devoted to the recent past. As men with this type of training fill posts in our veterinary colleges, and become the veterinary educators of the future, it is inevitable that fuller attention will

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be given to the totality of the training and education of students—who will become *comparative pathologists* in the fullest sense.

Summary

The development of veterinary pathology as a scientific discipline in its own right has been dependent upon: (1) the regular practice of post-mortem examination, (2) the publication of articles and texts clearly identified as standard bearers of the subject, and (3) the establishment of regular chairs of veterinary pathology occupied by properly trained and educated veterinary pathologists. It is suggested that the historical designation of *comparative pathologists* be kept in mind in the training of veterinary students, and that the full education of the veterinary pathologist requires some awareness of professional history.

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AN OCCURRENCE OF TOXICITY ASSOCIATED WITH PHENOTHIAZINE IN WEST AFRICAN CATTLE

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THE following report is, so far as the author is aware, the first record of phenothiazine toxicity occurring in cattle in Africa. Clare (1952) in his monograph on photosensitisation refers to cases of phenothiazine toxicity recorded in other countries by various authors.

This toxicity occurred at Katabu, in the Zaria Province of Northern Nigeria. The herd in which the cases occurred had been used for trypanosomiasis research, and consisted of three Zebu, eight N'dama and seven N'dama x Zebu cattle; the presence of trypanosomes in all the cattle except four of the N'damas had been noted previously. A complete history of this herd is recorded elsewhere (Chandler, 1952).

Two other herds at Katabu and two herds at Lumu (30 miles from Katabu) were also dosed with phenothiazine; these four herds each consisted of about 20 Zebu cattle and were used for trypanosomiasis research. Most of these cattle were protected by Prophylactic Antrycide and some were unprotected controls. Complete histories of these herds are recorded elsewhere (Unsworth and Chandler, 1952).

The phenothiazine which was used to control helminthiasis, consisted of 5-g. tabloids and was stored in tins, sealed with adhesive tape (i.e. not exposed to damp or light) and kept on a cool, shaded shelf. The instructions in the tin stated the dose for sheep to be 4 to 6 tabloids. No ox which subsequently showed toxicity symptoms was given more than 10 tabloids, i.e., never more than twice the dose for a sheep. None of the cattle dosed were heavy in calf except one N'dama which in any event did not show toxicity symptoms. The dose for cattle is quoted by a well-known pharmaceutical firm as being 40 to 60 g., i.e. equivalent to 8 to 12 tabloids.

The dose-rate adopted for the four Zebu herds was based on their body-weight (calculated by measurement formula); cattle of 100 kg weight were given 6 tabloids (30 g.), cattle of 300 kg. 10 tabloids (50 g.) and the intermediate weights dosed proportionately. The dose-rate for the mixed N'dama and Zebu herd was as follows: Large Zebu, 10 tabloids; medium-size Zebu, crossbreds and one N'dama, 8 tabloids; and the other N'damas, 7 tabloids.

One herd of Zebu at Katabu and one at Luma, dosed on May 31, showed no symptoms, and the other two Zebu herds at Katabu and Lumu, when dosed on July 7, also showed no symptoms. The mixed N'dama and Zebu herd at Katabu, dosed on June 3, showed no symptoms but when the latter herd was dosed on

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September 2 to 6, and given the same doses as in June, the following toxic effects were noted.

Toxicity symptoms occurred in one of the three Zebu cattle, and in four of the seven N'dama x Zebu cattle; trypanosomes had previously been detected in the blood of all the Zebu and crossbreds and the majority showed some loss of weight, but no other symptoms of trypanosomiasis. Toxic symptoms also occurred in four of the eight N'damas; trypanosomes had previously been detected on rare occasions in the blood of two of these four N'damas but none showed symptoms of trypanosomiasis.

The symptoms associated with phenothiazine toxicity commenced four to five days after dosing, and consisted of conjunctivitis, keratitis, weakness, inappetance and, in one case, photophobia. All cases which showed symptoms ended fatally, except in the case of one N'dama which recovered. Death occurred in one to four weeks and in one case eight weeks after dosing. The lesions of most note, observed at examination post-mortem, were hepatic congestion and hepatitis, nephritis and ecchymoses of the epicardium.

It is of interest that, using the same stock of drug throughout, different groups of cattle were dosed in the months of May, June, July and September. Toxic effects were observed in September only. (The same doses used in June with the same herd gave no symptoms.) Cows with or without trypanosomiasis, and of all three breed-groups, and of both sexes, suffered. Since the toxic effects occurred in September and not in the previous four months one may suggest that the herbage eaten by the animals, which was lush and abundant at this time of the rainy season, might be connected in some manner with the toxicity.

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"STAGGERS" IN SHEEP

A review of some of the relevant information

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Introduction

DISEASES of sheep in which incoordination of movement is a characteristic symptom may be associated with a variety of predisposing and actual causes. The following is a list of factors which, under certain conditions, render sheep liable to demonstrate "stagger" symptoms. It is not a complete list and does not include diseases in which an animal may suffer from an odd convulsion, or demonstrate an abnormal gait.

Factors which may render sheep liable to stagger.

Plants:

Lolium perennæ (rye grass).
Lolium perennæ with *Claviceps purpurea* (ergot).
Paspalum dilatatum with *Claviceps paspali* (ergot).
Echinopogon ovatus (rough-bearded grass).
Poa argentina (pampas grass).
Phalaris tuberosa.
Malva parviflora (marshmallow).
Lamium amplexicaule (hedge nettle).
Stachys arvensis (stagger weed).
Chelianthes tenuifolia (fern).
Notholæna distens (fern).
Hypericum perforatum (St. John's Wort.).

Chemicals:

Sodium chloride (excessive intake).
Cobalt (deficiency).
Calcium (deficiency).
Magnesium (deficiency).

Bacteria and viruses:

Clostridium tetani (tetanus).
Erysipelothrix monocytogenes (listerellosis).
Virus of louping-ill.
Virus of scrapie.

Parasites:

Œstrus ovis (nostril fly).
Coenuris cerebrates (sturdy or gid).

Unknown factors which occur in:

Daft-lamb disease (cerebellar atrophy).
Stiff-lamb disease (muscular dystrophy).
Twin-lamb disease (pregnancy toxæmia).
Swayback (cerebral demyelination).
Migram.
Rida.
An "obscure" disease.

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Plants providing wholesome and often attractive grazing, but causing Stagger symptoms under certain conditions.

M. parviflora, L. amplexicaule, S. arvensis.—These are toxic during early growth and when seeding. Symptoms demonstrable only when sheep are disturbed or given enforced exercise.

P. tuberosa, L. perennæ.—These are most toxic during early growth following first rains after a dry summer and when predominating.

L. perennæ with ergot.—These are most toxic during honey-dew sporulation stage in seed-head.

Plants usually avoided by sheep and eaten only when no other choice of food available.

P. dilatatum with ergot.—Although this grass is wholesome, it is avoided when the seed-heads are infested with ergot.

E. ovatus.—This is only toxic during its early growth.

P. argentina.—This is toxic at any time, especially for lambs.

H. perforatum, C. tenuifolia, N. distens.—These are always toxic.

The various forms of "stagger" are described briefly under headings associated with the causal factors.

M. parviflora, L. amplexicaule, S. arvensis (Seddon, H. R., 1937).

Enforced exercise for a few minutes or up to an hour induces a shivering attack, with back arched, nose poked out, a staggering gait, incoordination and hurried and difficult respirations. If pressed to go on, sheep throw themselves down, rest, get up and after a period of trembling and shivering of the leg muscles, go down again. They may die within two hours, or, if not unduly pressed, may walk for miles without ill-effects.

P. tuberosa (McDonald, I. W., 1942).

Symptoms usually appear after one to three weeks' grazing. The flock appears normal until disturbed and then the chief abnormality of incoordination is observed—a drunken gait—and the sheep cannot move quickly. The legs move stiffly, the hindquarters sway, there is a marked tendency to fall, followed by a vigorous but ineffectual effort to regain balance. There is restlessness, hyperexcitability and continuous twitching of the ears. The head is shaken from side to side or nodded and fine tremors occur in the musculature. When the sheep is confined in a small area it attempts to hide in corners, stamps its feet and repeatedly jumps about on all four feet. When driven, the animal soon becomes exhausted, lies or drops down, and its breathing is laboured and its heart pounding. After a period of rest the nervous symptoms disappear and the sheep rises to its feet and behaves normally. If, however, an animal is disturbed continuously, attacks become more frequent and severe and may even terminate in death. When the disease is noticed early the sheep are moved to a fresh pasture containing no phalaris and the nervous condition soon improves and complete recovery may occur after a few weeks. If the disease is not noticed until the chronic stage is reached, it is found that the animals

are affected much more severely, deaths are more frequent, and recovery is very slow after change of pasture. In this chronic type, exercise produces rapid onset of fatigue and the heart sounds may be audible at several feet. When down, there may be difficulty in rising and the animal crawls along. In walking, the hind limbs may be carried stiffly and under the body in an awkward gait.

Examination post-mortem reveals nerve tissue degeneration in the latero-ventral tracts surrounding the grey matter—a symmetrical lesion extending throughout the entire length of the spinal cord. Lee and Kuchel (1953) showed that cobalt-treated ewes did not develop symptoms comparable with those exhibited by untreated ewes; the latter were in good condition and none showed signs of the progressive wasting characteristic of cobalt deficiency.

L. perennæ (Hopkirk, 1935).

No symptoms are observable until the flock is moved and then sheep start running and prancing with heads held high. A sheep may stand swaying, with eyes rolling and then fall over unconscious with legs straightened, head back, eyes twitching and rolled back and body trembling. Relaxation occurs after a few seconds and consciousness returns; the sheep regains its feet and runs a few more yards and repeats the performance. Death rarely occurs and examination post-mortem reveals no abnormality. Animals left alone will recover slowly, but there is a danger of accident and they may get caught up in fences or be drowned.

L. perennæ (*hypomagnesæmia*) (Blumer, *et al.*, 1939).

Observed in lactating ewes and those in a state of advanced pregnancy, whilst grazing on lush pastures; and paddocked sheep following enforced exercise.

Symptoms develop within a few hours and death may occur within four to six hours or after two or three days' illness, unless the animal receives treatment. There is a general lassitude and disinclination to move, flatulence and little or no bowel motion. The animal shivers and shows a stilted gait, especially if forced to move. Often the sheep cannot stand or collapses after staggering a few yards. When recumbent, it lies on its sterum with hind legs straddled out behind and looks as if it had " dropped in its tracks." Its head is stretched out and rests on its chin, and often there is a discharge of fluid ingesta from the nostrils. Pneumonia often develops and the mortality rate may be more than 90 per cent. In some cases, sheep have recovered after being unable to rise for four days.

Enforced exercise will produce symptoms within 24 hours in animals separated from a flock in which no symptoms develop under normal grazing conditions.

Chemical analyses of the blood sera of affected animals showed a disturbance of the Ca/Mg balance; and treatment of the sheep with calcium-magnesium intravenouslyy provided an immediate recovery.

Post-mortem findings were confined to the bowel and were characteristic: the rumen and reticulum congested with sour food; the omasum contracted;

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cæcum distended with gas; jelly-like mucous exudate; congestion of mesenteric glands and vessels.

L. perennæ with Ergot (Hopkirk, 1935; Steyn, 1934).

Claviceps purpurea (ergot) occurs on rye, wheat, oats, barley and grasses. Its mature sclerotium, or resting stage, is seen as a dark violet to black spur, and may be found in the seed-heads of the living plant, in hay, or loose on the ground. Staggers occurs when the grass is not seeding but is short, and it is uncertain whether the sheep pick up the sclerotia from the ground. The weather conditions which are responsible for fresh growth of grass are also ideal for the germination of the sclerotia; this germination involves the ejection of minute spores (ascospores) from mushroom-like outgrowths and these spores are carried upwards by air convection. Should any grass flowers be open at this time, the spores come into contact with the embryonic seed, and this is the only stage at which ergot infection can be initiated. Six to eight days after the primary infection, a sticky exudate called "honey-dew" develops in which spores (conidia) are embedded; these spores drop to flowers lower on the stem and are carried to other plants by insects, moving animals and swaying grass stems. This is the stage of development which is considered to be harmful when ingested by animals. The feeding to sheep of rye grass ergot in the sclerotia stage produces ulceration of the stomach and tongue alone. The usual symptoms of ergot poisoning are malaise with depression, rapid loss of condition and uncertain, ataxis movements. Frequently one of the hindlegs is affected first. There are other symptoms which include gastro-intestinal irritation, necrotic stomatitis, gangrene and mummification of the extreme parts of the body, and uterine contractions. If serious poisoning occurs, the ataxia increases to clonic convulsions and finally paralysis of the respiratory centre.

P. dilatatum with Ergot (Hopkirk, 1935, 1936; Hindmarsh and Hart, 1937).

In pastures where paspalum is well established it has been noted that even in times of prolonged drought this plant has continued to produce an astonishing quantity of palatable green feed, while the younger pastures in which rye grass and clover predominate have long been parched and bare. A brown berry-like excrescence on the seed-heads of paspalum indicate the presence of the ergot *Claviceps paspali.* This fungus is most toxic when passing from its sphacelial to its sclerotial stage, which is usually during warm, moist weather. Animals avoid the seed-heads, except when they are hungry. It is probable that little difference exists in the chemistry of the two species of ergot: purpurea and paspali. At the beginning of the illness affected sheep appear normal but are easily frightened by sudden noise or movement. This is followed by a drunken appearance with irregular gait, difficulty in maintaining balance and inability to rise; tremors of the muscles of the trunk and limbs with waving of heads; laboured breathing with salivation; accelerated heart action with an imperceptible pulse. There may be digestive derangement. If the animal goes down, its legs may be outstretched behind and,

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being unable to rise, death ensues. Those that recover take weeks to regain weight and condition.

Examination of animals post-mortem show an excess of clear fluid of low cell content and under pressure beneath the meninges.

E. ovatus (Seddon and Carne, 1926).

This is a grass which is usually avoided by sheep. It is plentiful in the late winter and early spring when other grasses are scarce, but is only harmful up to the time it seeds. Lambs are susceptible and affected animals stand with arched back and head swinging gently up and down or from side to side. On movement there is a marked stiffness in action with very little flexion of the carpel and tarsal joints and the limbs are flung forward in a stilted manner. The animal stands with forelegs extended and hindlegs spread widely apart. In more severe cases incoordination of the legs causes the animal to progress by a series of bounds. Finally there is loss of equilibrium and the animal falls, often when turning, and usually on the same side, and lies with legs stiffened and is unable to rise. When down, there is convulsive struggling, sweating and rapid breathing, and the sheep may roar as if in pain. If allowed to remain quiet the animal recovers somewhat and after some minutes is able to get up but the stiffness of action and shaking of head persists. In the most severe type there is marked hyperæsthesia, and acute symptoms may be brought on by the sudden appearance of dog or man. If in falling, the animal's head points downhill, it may be unable to rise and death may occur. Death is usually the result of accident due to obstacles such as holes in the ground, fallen trees or drowning in creeks.

In chronic cases the animal walks stiffly and, if approached, it runs away in a curious manner, with the hindlegs being moved together although the forelegs trot. Emaciation is a general effect of the disease due to the animal being unable to travel and forage for its food. Removal of an affected sheep from the suspected grazing leads to an improvement in four days and complete recovery within a fortnight.

Examination post-mortem reveals no gross pathological changes.

P. argentina (Jones and Arnold, 1917).

This grass is toxic all the year round and is eaten only when the finer grass is in short supply following a long continued drought. Older animals, used to this grass, are seldom affected. Symptoms develop at variable times after two to 21 days of feeding. They are characterised by weakness, muscular twitching, irregular movements of the head, stiffness of the limbs and transient motor paralysis with accompanying spastic spasms when excited. There may be derangement of vision and conjunctivitis. Hyperæsthesia is evident when the animal is touched on the head. Some sheep may recover spontaneously. A complete change of diet effects a cure in two weeks.

Post-mortem lesions are not characteristic.

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H. perforatum (Cunningham, 1947).

This plant is generally found on waste land and grows in small isolated patches. It is eaten only when there is a scarcity of the finer grasses and most outbreaks occur in the late summer or autumn. Experiments with ground-up flowers and leaves of this plant have reproduced the disease. The illness is a photosensitisation and is characterised by varying degrees of erythema and hypersensitivity. In cases of extreme hypersensitivity, which only occurs at the active stage of photosensitisation, a light knock on the ear or head may precipitate a convulsion. An animal may run for some distance, jumping about with stiffened legs and then stand with sharply-arched neck, or stagger about and rear. When the sheep starts to cross water there is a reaction as soon as the front feet are wet, and the animal rears on its hindlegs and jumps about, threshing with its feet. If the whole body is immersed as in dipping or swimming, a sensitive animal threshes wildly with all four limbs for a few seconds and then extends its neck and body and the whole body immediately stiffens. Some sink at this stage whilst others continue the convulsions. Those removed from the water may lie for a while in a tonic convulsion, then rise and stagger rapidly around, falling down in convulsive seizures, only to rise again and repeat the performance. After three to four minutes the animal recovers and appears normal. The convulsions usually follow head immersion. When dipping takes place in hot, sunny weather, those sheep which have been in the shade for some while before the operation do not suffer so badly. Only those which have grazed on pasture containing *H. perforatum* show any symptoms. There is also an erythema of unpigmented skin and exposed areas with some pruritus, œdema, necrosis and scab formation with shedding. The whole of the head, the mid-line of the back, and sometimes the legs may be affected. Loss of condition occurs in ewes and lambs. The mortality rate is not high. No post-mortem lesions are reported.

C. tenuifolia (Gordon, 1936).
N. distans (Seddon, 1937).

The symptoms demonstrated when either of these ferns is eaten, which is usually when other food is scarce, are rather similar. There is a slow, stumbling gait and an apparent blindness, and the animal becomes semi-comatose and does not run away when it is approached.

No examinations post-mortem are reported.

Cause Unknown. Migram (Chesney, 1953).

This disease appears to be associated with a deficiency of moisture in the diet and the ingestion of salt water during hot, dry weather. Occasionally it occurs in the winter time during very dry, frosty weather. Symptoms are observed most frequently from July to September, especially when the grass (rye-grass mixtures) is very short. The condition clears up when fresh water is provided. The earliest sign is exhibited when the flock is made to move on or hustled. Several sheep will be noticed lagging behind in a depressed manner

and when these are stimulated they will attempt to hurry and then demonstrate one or other of the following types of nervous reactions :—

(1) Sudden opisthotonos with knuckling of the hocks, fetlocks and sometimes the carpal joints, and the animal sinks quietly to the ground. There is no struggling and if it is not disturbed it will remain in this position for a few minutes, when it will rise and walk with an unsteady gait, but without stiffness, for about 50 yards and then appear quite normal. If it is hustled in the slightest during this walk it will repeat the same cycle of symptoms.

(2) Trots with an increasingly stiffening gait, its hindlegs become quite stiff as if tethered and are dragged behind as the belly sinks to the ground, whilst the front legs continue to struggle forwards. When it realises the futility of movement it lies quietly until the seizure has passed and then it rises and walks almost normally to rejoin the flock. If it is hustled during this walk it will repeat the performance and sometimes finishes up on its side, dithering.

(3) Sudden knuckling of the wrists with emprosthotonos and complete somersaulting once or twice, or pleurothotonos with dithering of the updrawn hindlegs for a variable period up to one minute. It will then become normal and quietly join the others unless it is disturbed, when it will repeat the same symptoms.

A recovered sheep will often stand with its hindlegs fairly wide apart and sway its body for some minutes. Repetition of the symptoms at intervals during the day do not appear to have any marked detrimental effect, even if the illness lasts several weeks. The degree of hypersensitivity varied with individuals. Some needed hustling whilst others reacted to a piece of paper blown across the path. When left quietly alone, the sheep appeared normal in every respect, grazing, resting and chewing the cud. Only when it was very hot and dry would they congregate along the ditches and it was usually during the middle of the day that most symptoms were observed. Sometimes in descending a bank a sheep would slip, stumble, or splash the water with its foot and that would be sufficient to stimulate a seizure. It would fall into the water, enact its own peculiar symptoms, usually with its head under the water, and die of drowning. All the deaths were by drowning. A number of sheep were found dead in the ditches, their bodies resting on the bank and their heads in three inches of water. Except during a seizure, there was no panting and there was never any nasal discharge, vomition or diarhœa.

Blood examinations made on eight sheep suffering from the disease showed no abnormality regarding the chloride, calcium, magnesium or hæmoglobin content.

Examinations post-mortem on four sheep showed no abnormal conditions apart from water in the lungs. No histological or chemical examinations were made.

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Cause Unknown. "An Obscure Disease" (Hayston, Hindmarsh and Walker, 1941-2).

The symptoms of this disease suggest a functional disease of the cerebellum. The disease affects all ages and either sex. The sheep were kept in paddocks containing surface tanks watered by artesian bore drains. These drains ran through several paddocks which held seriously-affected flocks into paddocks where the disease did not occur. Drought conditions prevailed from April to October and mortality averaged 12 a day. The ground was quite bare in parts and where the natural food failed, supplementary food was given. By October the sheep were thin and debilitated. Deaths occurred in most paddocks whether the sheep were hand fed or the natural pasture lasted well. Variations of type or amount of rations did not affect the incidence. Toxic plants as etiological factors were eliminated because certain paddocks where the disease occurred were devoid of all vegetation. Chemical examinations of the water supply and food supplements and feeding of these to experimental sheep exonerated these as possible causes.

In the early stages there is an unsteady gait, incoordination of movement and the sheep lags behind the flock. Later, it stands with legs held stiffly and if moved on, it stumbles and falls. Sometimes the neck is held high with muzzle pointed upwards, and the head swaying from side to side. The sheep often rocks backwards and forwards owing to the violent respiratory efforts which are often stertorous and jerky, with slow inspiration followed by a long interval and then sudden expiration with exaggerated movement of the abdominal muscles. In other cases the respirations are rapid and regular. Later, the sheep drops on to its sternum and sits with head turned back to the flank, or with nose pointing vertically and the head and neck slowly moving from side to side in a steady waving movement. As the disease advances, the sheep rolls over on to its side and shows convulsive spasms. Fine muscular tremors are present throughout the illness. The head is retroflexed on to the spine and the jaw is parallel with it, and there is champing of the jaws and frothing at the mouth. When down, the legs are held out stiffly and if the animal is stimulated by a light blow or noise it shows tetanic-like convulsions. In the interval between convulsions the general musculature is more or less tense and hard. Death usually occurs within 48 hours of the start of the symptoms and there is no certainty that any affected sheep recovers. The animal is found dead as if from a tetanic convulsion and there is a profuse nasal discharge and the adjacent ground is deeply furrowed.

Examinations post-mortem shows the main lesion to be in the brain. The surface vessels are congested; the organ is very œdematous and often soggy with fluid; the ventricles and subdural spaces have more fluid than normal. There is no perivascular cuffing or cellular infiltration and no degree of demyelination. The Purkinge cells of the cerebellum show slight degenerative

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changes. The mucosa of the stomach and intestines is congested and all the lymph glands are enlarged. There is slight tubular degeneration in the kidneys. All tests for incriminating organisms, viruses and toxins are negative.

Blood serum shows normal levels of calcium/magnesium.

Cause Unknown. Pregnancy Toxæmia (B.V.A., 1955).

This is an acute metabolic disorder affecting ewes during the last few weeks of gestation. The disease occurs when there is a deterioration in the quality and quantity of the diet in the last two months of pregnancy. In mountain sheep being wintered on a very limited area of grass, the condition may arise in ewes carrying only one lamb; whereas in half-bred ewes carrying triplets, cases may appear when the diet in late pregnancy is not appreciably defective. The animal appears stupid and is disinclined to move from place to place or move any part of its body. The head is carried in an unnatural position; it may droop, or be held on one side, or it may be carried very high. The gait becomes staggering and uncertain, and later there is marked disturbance of vision and even apparent blindness. The appetite is lost almost completely and intestinal peristalsis is sluggish or entirely suspended. Fine tremors appear over the head and neck and may become general. Temperature is normal. In the course of one or more days the ewe is unable to rise; she lies quietly; sensibility is dulled; tympanites may be extreme. There is marked reduction in blood sugar, a fall in the alkali reserve and acetonæmia. The calcium values are within the normal range, but hypocalcæmia may coexist with this condition. Recovery often occurs spectacularly after lambing, but unless the illness appears immediately prior to lambing, the mortality rate probably exceeds 90 per cent. Death occurs in from one to six days. Examination post-mortem reveals fatty infiltration of the liver with a depletion of glycogen. There is no corresponding depletion in the liver of the lambs.

Cause Unknown. Muscular Dystrophy in Lambs (B.V.A., 1955).

This condition, known as "stiff lamb disease," is generally thought to be of nutritional origin and has been found to occur in particular when the ewes, during pregnancy, are on a restricted diet, as in severe winter. The administration of vitamin E may both prevent and cure the condition. It affects lambs from about one to eight weeks of age and is characterised by a stiff gait, a disinclination to run or walk, difficulty in rising or in maintaining the standing position, or inability to stand up or even raise the body from the ground. Recovery may occur spontaneously in mild cases. In severe cases death, probably caused by the myocardial lesion, may occur more or less suddenly, or within a few days. As a rule the course of the disease is more prolonged and complicating lesions are the cause of death.

Examination post-mortem show degenerative changes in the musculature (voluntary and myocardium). Sometimes this is widespread but may be restricted in extent and could be missed. Often the muscles of the hind limbs or the back are almost white in colour, and in some areas may be dull white owing to calcification.

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Cause Unknown. Daft Lamb Disease (B.V.A., 1955).

This is an hereditary congenital cerebellar atrophy. Most affected lambs are unable to stand but some may be able to stagger over short distances. Whilst unable to maintain its equilibrium, the lamb retains its power of muscular movements, defæcation, urination, suckling and tail-wagging. The incoordination is of a more disorderly nature than that of "swayback." Examination post-mortem shows no specific lesions.

Cause Unknown. Swayback (B.V.A., 1955).

An idiopathic nervous disorder of new-born and young lambs (born of healthy ewes), the outstanding symptom of which is ataxia. In some way this disease is linked with a trace element deficiency because the administration of small amounts of copper to ewes throughout the gestation prevents the disorder appearing in the lambs.

In certain affected areas, most cases show symptoms at birth, but in others, some cases do not show clinical signs until the lambs are two to six weeks old. There is a great difference in the degree of severity. All show ataxia; some are unable to stand or walk and lie helpless; some may rise with difficulty to collapse almost immediately; others walk with a straddled gait; mild cases show only a slight weakness of the hindquarters, particularly when hustled. The ataxia is a form of spastic paralysis. Many affected lambs are blind. The disease is non-febrile and remission does not occur. In severe cases, the period of survival varies, but the mortality is 100 per cent. Death is often due to intercurrent infections. pneumonia, etc., while many die from neglect, exposure or marasmus. The most severe cases are those which show flagrant symptoms at birth. The mildest cases, in which symptoms may not be observed until some weeks after birth, may show only slight unsteadiness when walking, and if such animals are allowed to survive and are bred from later they may give birth to healthy lambs.

Examination post-mortem shows the main lesion to be in the brain. The amount of destruction appears to be in proportion to the severity of the symptoms, and the cerebral white matter may be completely liquefied, destroyed and cavitated, leaving a thin sheet of grey matter on the outside which, when punctured, causes the brain to collapse.

The disease processes undoubtedly originate in the fœtal lamb in the latter part of the gestation period.

Cause Unknown. Rida (Sigurdsson, 1954).

A chronic encephalitis, possibly caused by a filterable virus with a vector involved. It is considered contagious and some unknown environmental factor of fundamental importance is suspected as playing a part in epidemics.

Incubation period may last for eight to 18 months. The illness starts very gradually with an uncertain gait or slight tremor of the lips. It runs a protracted course and does not affect the general health until the late stages. Several weeks to several months usually elapse after the first symptoms until the animal becomes prostrate and dies. As far as is known, recoveries do not

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take place. Symptoms consist of spastic movements, incoordinated gait and tremors, indicating lesions of the extrapyramidal motor system. Temperature is normal and there are no anatomical changes outside the central nervous system. In advanced stages, the animals do not show the normal interest in food and water and they lose condition. They move with a wobbly gait and gradually become dehydrated and emaciated. Towards the end they lie on their sides with limbs outstretched and, although no individual muscles or groups of muscles appear to be paralysed, the animals are unable to move, and usually die in a stuporous state.

Scrapie (B.V.A., 1955).

There is evidence that this disease is due to a filterable virus, located in the brain, spinal cord and spleen, and that the infection occurs congenitally.

The early symptoms are nervousness, apprehension and over-excitability. Fine tremors over the head and neck cause slight but very rapid nodding. If rounded up the sheep become tense with excitement, and rapid muscular tremors, particularly affecting the thighs and flanks, are evident. The head and neck are carried high and sometimes stiffly; the facial expression is staring and fixed; the ears often assume an unnatural position; and often grinding movements of the teeth are evinced. Thirst is a frequent symptom. There is no diarrhœa, but if the animal be excited, quantities of fæces and urine may be passed involuntarily at short intervals. The fleece becomes lighter in colour and loses its lustre. The bleating is somewhat feeble, husky and tremulous. Appetite and temperature remain normal.

The most marked clinical feature is the generalised pruritis, which is a constant torment and probably the factor behind the progressive emaciation and weakness.

Often various motor nervous disturbances are manifested, such as aberration of gait, epileptiform convulsions and paralysis of the hindquarters. Many cases also exhibit erratic and blundering movements and, when made to trot, a number may show a peculiar gait in a high-stepping action of the forelegs, with or without "rolling" or galloping movements of the hindlegs. Often the hind hooves are trailed. When hustled or exerted, the sheep, after galloping for a few yards, may develop convulsive seizures suddenly. These are followed immediately by a varying degree of coma, but after lying quietly for some little time, consciousness returns and a condition of relative normality returns. Sometimes death occurs during a convulsion, but usually it follows six weeks to six months illness. The disease is seldom observed in sheep under 18 months of age. No specific curative or preventive treatment is known.

Examination post-mortem reveals no gross pathological changes.

Louping-ill (B.V.A., 1955).

This is an epidemic encephalomyelitis, characterised by acute fever, malaise and cerebellar ataxia. It is caused by a filterable virus which is transmitted through the medium of the tick, Ixodes ricinus, and is, therefore, confined to tick-infested areas. The incidence of the disease in acclimatised sheep is high,

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but many show no symptoms other than those associated with fever. In others, after about 5 days of dullness and fever, the virus may attack the cells of the central nervous system, and this may be followed by symptoms of excitability, tremor (especially of the head), muscular spasms and irregularity of gait; the balance may be affected and the sheep become unable to stand; in the later stages paralysis of one or both hind limbs may develop. Some sheep may recover during the early phase without developing nervous symptoms. Mortality is greatest in the young—about 4.0 per cent in yearling sheep. The mortality in non-acclimatised sheep, introduced to louping-ill pastures, may exceed 60 per cent.

Examination post-mortem reveals no gross pathological changes, but there are degenerative changes either in the Purkinge cells of the cerebellum or in the large nerve cells in the grey matter of the spinal cord, together with changes representative of encephalomyelitis.

No specific curative treatment is known, but if affected sheep are kept in sheltered, quiet surroundings and carefully nursed, the chance of recovery is definitely increased. The disease can be effectively prevented by the use of vaccine prepared from the virus.

Listerellosis (B.V.A., 1955).

This disease, which is an encephalitis, is caused by Erysipelothrix monocytogenes. The symptoms are dullness, blindness, circling, leaning and opisthotonos; death occurs in about five days. There is no monocytosis in sheep.

Examination post-mortem reveals perivascular cuffing, and often some purulent foci in the brain and œdema of the meninges may be noted.

Tetanus (B.V.A., 1955).

The muscular spasms characteristic of this disease are the result of the action on the nervous system of the toxin produced by Clostridium tetani when this organism is present in wounds. The condition occurs most frequently in lambs, but does occur in ewes after lambing.

When lambs are mildly affected the only symptom may be muscular stiffness, and such cases generally recover. In more acute cases the animals become recumbent, the jaws may be more or less fixed, while tetanic muscular spasms may throw the body into a state of rigidity, with the limbs extended stiffly, the back hollowed and the head retracted. The symptoms are exacerbated by lightly tapping the animal. When these acute symptoms are in evidence recovery cannot be expected.

Examination post-mortem reveals no specific lesions.

Parturient Hypocalcæmia (B.V.A., 1955).

This condition, commonly called " lambing sickness," is an acute neurosis of the lactating and pregnant ewe. It is believed to result from a derangement of the function of the calcium-regulating mechanism, so that the calcium content of the blood and soft tissues is suddenly and profoundly decreased. It is characterised by sudden onset and a short, rapid course which frequently terminates in death unless treatment be applied promptly.

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In the primary phase of the attack the symptoms are those of extensor tetany of the limbs, accompanied by considerable excitement and general distress; these symptoms may. however, be slight and transient, and they are usually very quickly followed by the comatose phase in which the ewe lies completely prostrate and insensible. A fatal termination may result within 24 hours. If acute tympanites develops during coma, asphyxiation may be the immediate cause of death. In the pregnant ewe many cases occur when the animal has been subjected to fatigue or over-exertion.

On examination post-mortem no lesion has been recorded. The curative response to calcium therapy is immediate and complete.

Pining (B.V.A., 1955).

This is a progressive debility, accompanied by anæmia and emaciation, and is due to a B12 avitaminosis associated with a cobalt deficiency of the soil. Lambs grazing on such deficient pasture do not thrive, and shortly after weaning begin to develop symptoms. The onset is frequently insidious. The animal is dull, and the fleece becomes dry, lustreless and broken; the visible mucous membranes, especially the conjuctiva, are pale; physical condition is gradually lost, the eyeball becomes sunken and there is often a profuse discharge from the eyes. In lambs growth is retarded, and they soon present a stunted, unthrifty appearance; thereafter the anæmia and emaciation progress, the gait is stilted and somewhat incoordinated, and finally, as the result of extreme weakness, the animal is unable to rise. In many instances the symptoms are aggravated by a superimposed nematode worm infestation, and it has been shown by experiments that cobalt-deficient sheep are more susceptible to helminthiasis than normal sheep. In many instances the condition presents itself in a much less acute form and is then considered more of a low grade of health than a definite disease. If therapeutic measures are not adopted the mortality rate in the acute form may be very high. On some farms it may reach 20 per cent and, apart from actual mortality, the physical condition of the majority of the flock is much impaired.

Examination post-mortem reveals anæmia and emaciation and occasionally a fatty degeneration of the liver.

The weekly administration of cobalt chloride to the sheep or the application of cobalt sulphate as a top dressing to the land, constitute the main forms of treatment.

Salt Poisoning.

In ordinary dehydration there is a marked loss by the animal of fluid containing solutes. It is a common sequel to prolonged vomition, diarrhœa, diuresis or profuse sweating and there is an increase in the concentration of sodium and chloride in the extracellular fluid with a consequent withdrawal of water from the cells. This is further aggravated following excessive salt intake and the alteration in the osmotic pressure of the body fluids is generally accepted as the cause of the nervous symptoms in salt poisoning.

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This condition may follow actual drenching with a salt solution, or the ingestion of saline waters when the animal is abnormally thirsty; or accidental contamination of food with salt; or the excessive intake of salt following a prolonged absence of the chemical from the diet.

The symptoms are an excessive thirst, a tendency to vomit, loss of appetite, diarrhœa, nervous twitchings, a staggering gait suggestive of marked weakness, muscular spasms, and sometimes blindness. The only treatment is an ample supply of fresh water.

Cœnuriasis (B.V.A., 1955).

This disease is insidious in its onset and runs a chronic course. The affected sheep remains in apparent health until some four to six months after infection. Up to five per cent of the sheep may become affected more or less simultaneously. When the cyst is located in the common site—in a cerebral hemisphere—the sheep holds it head to the affected side and tends to walk in a circle. They may be blind in the eye on the other side. If the cyst is situated in the anterior part of the brain, the sheep usually steps high; if it presses on the cerebellum, a hypersensitive condition and a jerky or staggering gait are common. The affected animal tends to wander from the flock and to become indifferent to food and water; frequently sight is affected and the facial expression is unnatural; grinding of teeth, salivation and complete loss of balance are also observed. A progressive paresis of one or both hind legs is seen in those cases in which the cysts are situated in the lumbar region of the spinal cord. When the cysts are present on the convexity of the cerebral hemispheres, they sometimes produce pressure atrophy of the skull, which becomes soft and yields easily to digital pressure over the site.

An intradermal test, using fluid from sturdy cysts, has been developed as an aid to diagnosis.

Surgical treatment is the only remedy, but as a rule the sheep should be slaughtered.

Nostril Fly (B.V.A., 1955).

Symptoms of this disease may resemble those seen in some cases of cœnuriasis. It occurs most frequently in July to August and when the infestation is heavy, the sinuses become filled with pus. There is a characteristic snorting or snuffling and a glairy or purulent nasal discharge which helps in the diagnosis of the disease.

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SUMMARY OF CHARACTERISTIC FEATURES

	Ins	H/S	E/E	Pru	Coma	L/C	N/F	F.	N/T/D
M. paryiflora			▪				▪	▪	
L. amplexicaule			▪				▪	▪	
S. arvensis			▪				▪	▪	
L. perennae			▪				▪	▪R	
P. tuberosa			▪			▪	▪	▪	▪
P. dil. with ergot		▪	▪				▪	▪	
Migram	▪	▪	▪				▪	▪A	
Obscure disease		▪	▪			▪		▪	▪
Swayback	▪/-		▪/-			▪	▪	▪	▪
Scrapie	▪	▪	▪	▪		▪	▪	▪	
Rida	▪		▪/-		▪L	▪		▪	▪
P. argentina		▪					▪	▪	
H. perforatum		▪		▪		▪	▪	▪R	
R. ovatus		▪				▪	▪	▪A	
Tetanus		▪L					▪	▪	
Part. hypocalc.		▪E			▪L			▪	
Preg. toxaemia	▪				▪	▪	▪	▪	
C. tenuifolia					▪E		▪	▪	
N. distens					▪		▪	▪	
L. perenn. ergot					▪		▪	▪	
Louping-ill					▪E		▪	▪	▪
Pining	▪				▪L	▪		▪	
Listerellosis					▪			▪	▪
Musc. dystrophy						▪	▪	▪	

Interpretation: Ins: Insidious in onset. H/S: Hypersensitivity. E/E: Enforced exercise. Pru: Pruritis. Coma: Comatose or semicomatose. L/C: Loss of condition. N/F: Non-Fatal. F: Fatal. N/T/D: Nervous tissue degeneration. "/-: May or may not be. A: Accidental. E: Early. L: Late. R: Rarely.

A STUDY OF beta-HÆMOLYTIC STREPTOCOCCI IN THE FADING PUPPY IN RELATION TO CANINE VIRUS HEPATITIS INFECTION IN THE DAM

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Short title: beta-Hæmolytic streptococci in the fading puppy.

In spite of continued investigation the widespread occurrence of the "fading puppy" remains a major problem for dog breeders. In a typical case the clinical picture was as follows:—

The bitch, apparently perfectly healthy, whelped to time without trouble or assistance. The puppies appeared to be strong and able to suckle during the first 24 hours of life. Several were then noticed to reach the bitch to suckle but their heads swayed slightly and they obviously found difficulty in finding the teat. Although they suckled, the vigour which had been shown previously was not so noticeable. Their backs became more arched than usual. The fore-legs "paddled" the mammary glands only weakly and the hind limbs did not exhibit their previous strength or "push." The puppies soon tired, and instead of moving away from the mother with a full stomach to sleep, the hold upon the teat gradually loosened and they rolled over and gave vent to faint cries. The bitch at this stage, instead of licking and nosing the puppy, showed less interest and refused to assist in any way. These signs gradually became worse during the next 24 hours. The puppies, being hungry, cried in a characteristic manner (weak and high pitched) but the mother, rather than assist, not only appeared to lose interest in the offspring but tended to push them away from her into a corner of the whelping box. The puppies died within a few hours unless they were kept warm and held to the teat to suckle. They seldom seemed to sleep as healthy puppies do, i.e. curled on their sides and giving those occasional twitches of the body which are so often a sign of good health. The puppies were rather restless with hind flaccid limbs stretched out behind. The tongue was pale. The abdomenal skin was pale and quickly lost its "tone." All the puppies were affected in the same manner and died within 96 hours.

The role of beta-hæmolytic streptococci in this syndrome has been examined by many earlier workers including Stafseth, Thompson and Neu (1937), Hare and Fry (1938), Minett and Ellis (1940), Hare (1946). and Laughton (1948), and although it has been proved to be the cause in many cases, it does not account for all of them by any means. When reviewing this syndrome, a number of other possible causes must be considered. Among these are general kennel management, nutrition, heredity factors including possible incompatible blood groups of the sire and dam, and infection with other bacteria and viruses. From investigations already made the micro-organisms most likely to be significant are beta-hæmolytic streptococci and the virus of Canine Hepatitis (Rubarth's Disease). From post-mortem examinations made by ourselves it would appear

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that certain strains of *Bacterium coli* might also be a contributory factor, but so far no experiments have been done to prove this theory.

It is almost certain that in any particular litter showing fading puppies, more than one of the above factors may be concerned, one being contributory to the other, and each must be considered according to its significance. The aim of the present investigation was to determine the incidence of dual infection with beta-hæmolytic streptococci and Canine Virus Hepatitis, and if possible assess their significance in puppy mortality.

Materials and Methods

In order that the observations made should be strictly applicable to conditions "in the field," only carcases submitted for diagnostic work were included in the survey. Of these, 138 were received from 24 counties in England and two in Wales, during a period of 33 months. The puppies varied in age from one to eight days.

Bacteriology

At post-mortem, examination swabs were taken from thorax and lung tissue and from the abdominal cavity, and from any other organ when necessary. Blood was withdrawn by heart puncture. Swabs and blood were cultured in duplicate on 5 per cent horse blood agar plates, one of which was incubated anærobically, and on McConkey's medium. The heart-blood was cultured also and in digest broth. All cultures were incubated at 37° C. and if sterile or showing only poor growth, after 24 hours, they were re-incubated for a further 48 hours.

All beta-hæmolytic streptococci isolated were subcultured to serum broth (20 per cent v/v), from which they were tested for biochemical activity in Hiss's serum water sugars, hydrolysis of sodium hippurate according to the ferric chloride test, final *pH* in glucose broth and virulence for mice. They were tested for the production of soluble hæmolysin by the method of Hare and Colebrook (1934), and subcultured to glucose broth (0.25 per cent w/v) for determining Lancefield's groups. This was done by layering the polysaccharide extract over specific group antiserum prepared in rabbits from known strains and also that produced commercially in capillary tubes.

All strains isolated were tested for sensitivity to penicillin, streptomycin, Aureomycin, Terramycin and chloramphenicol using a ditch plate technique.

Histology

The general appearance at post-mortem examination was noted, and samples were taken from liver, lung, kidney and spleen. These were kept in 10 per cent formol saline until the results of the bacteriological examination were known, and if beta-hæmolytic streptococci had been isolated or if the general appearance had suggested Canine Virus Hepatitis, histological examinations were made. The tissues were prepared by the normal methods of dehydration, embedded in paraffin wax and cutting of sections. In some cases serial sections were cut.

For routine examination the sections were stained with hæmatoxylin and eosin. Other additional methods used included the Picro-Mallory stain for hyalin changes, Periodic Acid Schiff's—counterstain phosphotungstic acid Orange G for eosinophilic properties, Prussian Blue for hæmosiderin, silver impregnation for reticulum, Landrum's stain for intranuclear inclusion bodies and Gram's stain for organisms.

In some cases, however, post-mortem decomposition was so far advanced by the time the carcases were received that histological examination was not possible, even on those from which it was still possible to isolate beta-hæmolytic streptococci.

When positive results were obtained from the puppy carcases, requests were made to the veterinary surgeons in charge of the cases for a sample of milk and clotted blood from the dam, and when these were obtained they were examined by the complement-fixation test for antigen and antibody for the virus of Canine Hepatitis according to the method of Larin (1951).

No attempts were made to establish either or both infections experimentally in bitches with litters, partly because of the artificial conditions which would inevitably be imposed, and partly because attempts to do so Stafseth (1940), and Laughton and Larin (1954), had been unsuccessful.

Results

Examination of the carcases post-mortem showed that in those from which beta-hæmolytic streptococci were isolated there was a generalised congestion, with petechial hæmorrhages on the pleura, peritoneum and intestine. In a few

TABLE I

138 puppies examined for streptococcal infection in relation to virus hepatitis in the dam.

Group I	Group II	Group III	Group IV
Streptococci isolated Virus not proven	Streptococci not isolated Virus established	Streptococci isolated Virus established	Streptococci not established Virus not proven
18	23	4	93

cases patches of œdema were found on the lungs. Carcases of puppies from dams in which Canine Virus Hepatitis was confirmed, also showed a generalised congestion, but this was most prominent in the liver. This organ was very dark in colour and the cut surface was pulpy and suffused with blood. In some cases the lungs showed patches of œdema similar to those found in cases of beta-hæmolytic streptococcal infection. Petechial hæmorrhages were not seen but the intestine showed patches of hæmorrhagic infiltration.

For purposes of comparison the puppies examined were arranged in four groups as shown in Table I.

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Bacteriology

From Table I it can be seen that beta-hæmolytic streptococci were isolated from 22 (15.9 per cent) of the 138 carcases examined. As far as could be determined only one strain was present in each carcase. The biochemical activity of these strains is shown in Table II, and their other characteristics investigated in Table III.

TABLE II

Biochemical activity of 22 strains of hæmolytic streptococci isolated from dogs.

Case No.	No. of pups	Glucose	Lactose	Dulcitol	Saccharose	Mannitol	Maltose	Inulin	Trehalose	Sorbitol	Glycerine	Mannose	Hydrolysis of sodium hippurate
153	1	+	+	-	-	-	+	-	+	-	weak +	-	-
226	2	+	+	-	+	+	+	-	+	weak +	+	+	+
264	1	+	+	-	+	-	+	-	+	-	+	+	-
268	1	+	+	-	+	+	+	-	+	-	+	+	+
308	2	+	+	-	+	-	+	-	+	-	+	+	+
353	1	+	+	-	+	-	+	-	+	-	+	+	+
448	1	+	+	-	+	+	+	-	+	-	+	+	+
567	2	+	+	-	+	+	+	-	+	weak +	+	+	+
601	1	+	+	-	+	+	+	-	+	+	+	+	-
865	1	+	+	-	+	+	-	-	+	-	-	+	-
1139	1	+	+	-	+	-	+	-	+	-	-	+	-
1166	2	+	+	-	+	-	+	-	+	-	-	+	-
1196	2	+	+	-	+	-	+	-	+	-	-	+	-
1262	3	+	+	-	+	+	+	-	+	-	-	+	-
1436	1	+	+	-	+	+	+	-	+	-	-	+	-

\+ fermentation of sugar or hydrolysis of sodium hippurate.
— sugar not utilised, sodium hippurate not hydrolysed.

Although it is recognised that classification of streptococci according to fermentation reactions (Table II) is not entirely satisfactory, the strains examined showed a predominance of trehalose fermenters. All 22 strains fermented this sugar; one only fermented sorbitol to an appreciable extent and four other strains attacked it feebly. This result would suggest that most of the

strains infecting the puppies were of human rather than of bovine origin. The fact that 12 of the 22 strains, or 53.6 per cent, fermented mannitol indicated that approximately half of these strains were enteric rather than salivary types. Sodium

TABLE III

Some further characteristics of 16 strains of hæmolytic streptococci isolated from dogs.

Case No.	No. of pups	Lancefield group	Soluble haemolysin	Liquefaction of gelatin	Final pH	Virulence for mice
153	1	M	none	-	7.0	none
226	2	L	present	-	6.0	none
264	1	G	present	-	6.5	none
268	1	G	none	+	5.5	none
308	2	B	present	-	6.0	none
353	1	M	present	-	6.5	virulent
448	1	M	present	-	6.0	none
567	2	M	none	-	6.0	none
601	1	G	present	+	6.0	virulent
865	1	M	present	-	6.0	none
1139	1	L	none	-	6.0	none
1166	2	G	none	-	6.0	none
1196	2	G	present	-	6.8	virulent
1262	3	G	present	-	5.5	none
1436	1	G	none	-	5.5	none

hippurate was included in the tests to detect common mastitis strains, but in view of the other observations described, this test seems unlikely to be of value.

Table III shows that strains giving the precipitin reaction of Lancefield's Group G type were most prevalent, 11 of the 22 strains examined belonging to this group. Six other strains belonged to Lancefield's Group M, and the remainder were fairly equally distributed. two in Group B, and three in Group L.

Soluble hæmolysin was produced by 14 of the 22 strains isolated, apparently without specific relation to their Lancefield groups. Two of the positive strains belonged to Group B, seven to Group G, two to Group L, and three to Group M.

Only two strains, both belonging to Group G, showed proteolytic activity by liquefying gelatin.

The final *pH* of the cultures in glucose broth varied from 5.5 to 7.0, without any apparent relationship to the Lancefield group of the strain.

The strains as a group did not show a high degree of virulence for mice by the subcutaneous route, only one of Lancefield's Group M and three of Group G strains proving fatal in 48 hours, with subsequent recovery of the strain from the mouse tissues.

All 22 strains were sensitive to a greater or lesser degree to Aureomycin, Terramycin and chloramphenicol. All but one strain, isolated from two puppies which were litter mates, were sensitive to streptomycin and all but three strains were sensitive to penicillin. Two of these strains were Lancefield's Group G, while the third belonged to Group M. They differed from the strain which was resistant to streptomycin and which belonged to Lancefield's Group B.

Histology

Histological examination was carried out on liver, lung, spleen and kidney of 12 carcases from nine cases in Group I (Table I), of 13 carcases from 10 cases in Group II and of four carcases from two cases in Group III. The changes observed are described below and shown in tabular form in Table IV.

Group I.

Liver.—In the majority of cases the most prominent change was dilatation of the sinusoids and blood vessels which were packed with red blood corpuscles. In three cases hæmorrhages were noticed but the walls of the blood vessels did not show damage. The nuclei of the parenchymal cells showed regressive changes in the form of rhexis. Histiocytic reaction was not prominent and was found only in two cases. Disappearance of the parenchymal cells was found only in one case and it occurred in the form of small foci without special location. The Kupffer cells did not show any changes nor did the reticulum pattern. The latter in some cases (five) was only partly lost.

Lungs.—Inflammatory changes were observed where alveolar capillaries and bronchial capillaries and arterioles were distended and packed with blood. The bronchial epithelium showed desquamation and proliferation in some cases, but inflammatory exudate was present in the lumen of the bronchi only in one case. An increased amount of connective tissue was noticed in five cases. Rupture of the alevolar walls appeared in patches and in two cases only.

Kidneys.—Hyperæmia of the cortex with increase in connective tissue was found in the majority of cases. The glomerular tufts showed a high cellularity with enlargement in some cases. The convoluted tubules shcwed disorganisation in all cases except one involving two puppies. In some cases the lining of the tubules showed swelling, granular changes and vacuolation.

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Spleen.—The most striking change was œdematous infiltration of the white pulp and its disappearance. Regressive changes were seen in the nuclei. In the red pulp there was an increase in the amount of blood present, but this change was not prominent.

TABLE IV

Histopathological changes found in 29 puppy carcases.

Organ	Histopathological Change	No. of cases showing change		
		Group I (12 cases)	Group II (13 cases)	Group III (4 cases)
LIVER	Intranuclear inclusion bodies	0	0	3
	Swelling of parenchymal cells	3	9	1
	Granularity of cytoplasm	2	6	1
	Eosinophilic properties	5	3	0
	Disappearance of parenchymal cells	0	1 (foci)	0
	Histiocytic reaction	3	8	0
	Cellular infiltration	4	9	4
	Enlargement of Kupffer cells	0	0	0
	Distortion of reticulum pattern	4	6	0
	Loss of reticulum pattern (partial)	7	6	0
	Perivascular hyaline changes	2	9	3
	Dilatation of sinusoids	8	9	4
	Haemorrhages	5	1	0
	Regressive changes of the nuclei	9	6	4
	Dilatation of blood vessels	10	9	4
LUNGS	Dilatation of alveolar capillaries	8	8	3
	Irregularity of the alveolar epithelium	9	8	4
	Proliferation of connective tissue in the alveolar walls	4	9	4
	Inflammatory exudate in the bronchi	2	1	3
	Desquamation of bronchial epithelium	5	3	0
	Proliferation of bronchial epithelium	4	1	3
	Distension of bronchial capillaries and arterioles	12	12	4
	Increased amount of connective tissue	6	11	4
	Rupture of alveolar walls (patches)	3	6	3
KIDNEYS	Hyperaemia of the cortex	9	0	4
	Hyperaemia of the medulla	3	2	1
	Red blood cells in glomeruli	6	9	4
	Enlargement of the glomerular tufts	2	5	4
	Cellularity of the glomerular tufts	12	9	4
	Adhesions between tufts and Bowman's capsules	5	8	4
	Disorganisation of convoluted tubules	10	13	4
	Swelling of tubular lining	3	9	3
	Granularity of tubular lining	3	9	0
	Vacuolation of epithelial cells	8	7	1
	Increased connective tissue in cortex	10	3	4
SPLEEN	Increased blood content in red pulp	7	12	4
	Increased blood content in white pulp	4	2	3
	Haemosiderin	2	1	0
	Diminution of white pulp	0	4	0
	Disappearance of white pulp	8	3	4
	Regressive changes in the nuclei	6	9	4
	Oedematous infiltration of red pulp	4	3	0
	Oedematous infiltration of white pulp,	10	7	1

Group II.

Liver.—In addition to the dilation of the sinusoids and blood vessels found in Group I, the parenchymal cells were swollen and their cytoplasm was granular, with eosinophilic properties in some cases. The foci situated centri-

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lobularly where the parenchymal cells disappeared were seen only in one case. A histiocytic reaction and perivascular cellular infiltration were seen in the majority of cases.

Lungs.—There was a distention of the bronchial capillaries and arterioles and an increase of connective tissue in nearly all cases. One case showed inflammatory exudate in the lumen of the bronchi with proliferation of the bronchial epithelium. Rupture of the alveolar walls occurred in patches and was noticed in 50 per cent of the cases.

Kidneys.—An increased volume of blood was seen in the glomeruli which also showed enlargement and adhesions with Bowman's capsule. The cellularity of the glomerular tufts was marked. In all cases there was a disorganisation of the convoluted tubules, with swelling, granularity and vacuolation of the tubular lining.

Spleen.—In the majority of cases there was a hæmorrhagic infiltration of the red pulp and an oedematous infiltration of the white pulp. In some cases the white pulp disappeared; in others it was diminished. Regressive changes of the nuclei were present.

Group III.

Liver.—Changes found in the liver were most prominent in this group. Perivascular infiltration, with dilatation of the sinusoids and blood-vessels, which were packed with blood, were seen in all cases. Regressive changes in an advanced stage were noted in the nuclei of the parenchymal cells but none of the parenchymal cells had disappeared. Intranuclear inclusion bodies were found in three puppies, the cytoplasm of the cells showing granularity and eosinophilic properties. A perivascular hyaline deposit was observed in these three puppies.

Lungs.—In the three puppies showing intranuclear inclusion bodies in the liver there was œdema with rupture of the alveolar walls, and an irregularity of these walls with proliferation of the connective tissue. In the fourth case in this group, the alveolar and bronchial capillaries and the bronchial arterioles were distended and packed with blood.

Kidneys.—Hyperæmia of the cortex with an increase of connective tissue was present in all cases. The glomeruli were cellular, enlarged and showing adhesions to Bowman's capsule. The convoluted tubules were disorganised and their lining swollen.

Spleen.—An increased content of blood was present in the red and white pulp. Regressive changes of the nuclei were observed.

Complement-fixation tests for antibody and antigen of Canine Virus Hepatitis were made. In 19 cases, involving 27 puppies, it was possible to obtain samples of milk and blood from the dam, and in one case, blood from the sire also.

Complement-fixation tests gave clearly-defined results on all the sera tested for antibody and on samples of the puppies' livers tested for antigen. Using the same technique, in two of the 18 cases (11.1 per cent), a positive result was obtained for virus antigen in the dam's milk, and a weakly positive result in a

third case. Blood from the dams showing a positive reaction in the milk also showed a positive reaction for antibody, while that from the dam giving a weakly positive milk reaction gave a weakly positive one for serum antibody. All tests tests for antigen in the puppy livers were negative except in one case in which intranuclear inclusion bodies had been seen in smears. and this liver showed a

TABLE V

Complement-fixation test for Canine Hepatitis Virus or its corresponding antibody in 19 bitches and their litters.

No. of cases	No. of puppy carcases	Dam's serum	Dam's milk	Puppy's liver
1	1	Positive	Positive	Negative
1	1	Positive	Positive	Negative
1	1	Weakly Positive	Weakly Positive	Negative
1 (sire's serum negative)	1	Positive	Negative	Weakly positive (inclusion bodies present)
	1	Positive	Negative	Negative
3	1 each	Positive	Negative	Negative
9	1 each	Negative	Negative	Negative
1	2 each	Negative	Negative	Negative
1	3 each	Negative	Negative	Negative
1	5 each	Negative	Negative	Negative
Controls 3	8 altogether	Negative	Negative	Negative

weakly positive reaction for antigen. In this case, in which the blood of the dame was positive, the blood of the sire was also available and found to be negative when tested on two separate occasions.

Three healthy bitches with litters were available for use as controls. Tests on blood and milk from the bitches and on the liver of several puppies from them all gave negative reactions for Canine Virus Hepatitis antibody and antigen.

These observations are summarised in Table V.

Further study of the 93 puppies placed together in Group IV is not relevant to the subject matter of this paper and therefore will not be described.

Discussion

As stated in the text (Table I), the 138 puppy carcases examined were divided into four groups of which the first three were investigated in detail.

The 22 strains of beta-hæmolytic streptococci isolated were distributed in Groups I and III in the proportion of 18 to 4, all those in Group III belonging

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to Lancefield's Group G. The preponderance of Group G strains is in agreement with the observations of Laughton (1948), and also of other workers, who found that Group G strains predominated in neonatal deaths in puppies. Laughton observed that Group M strains occurred most frequently in healthy dogs, but in the present study they were isolated almost as frequently as Group G strains. While these differences might be due only to changes in time and locality, it is nevertheless possible that if, as Laughton suggests, only Group G strains are of pathogenic significance to the dog, the Group M strains were merely incidental and played no part in the death of the puppy from which they were isolated. Unfortunately, it was not possible to obtain swabs from tonsils and vagina of the dams of the puppies in order to determine if the bitch was carrying a similar strain. In view of the very high carrier rate established for all types of streptococci in the dog, such as 92 per cent by Pilot (1936), 73.3 per cent by Laughton (1948) and 87.5 per cent by one of us (M.E.D.) in 1954, it would seem quite possible that a number of strains could be isolated from dead puppies without being of pathological significance.

There was no apparent difference in the types of streptococci isolated from the two groups as indicated by their biochemical characteristics. The fact that all strains fermented trehalose and a few fermented sorbitol as well, is in agreement with an observation of Minett (1935) who found that three of six strains tested fermented both sugars, with a concurrent lowering of the final *pH* of the medium to about 6.0.

A noticeable feature of the analysis according to Lancefield's grouping technique is the diversity of the groups now appearing. in contrast to the findings of Minett and Ellis (1940) and referred to by Stableforth (1938), who found a majority of Group G strains.

During the histological examinations differences between the three groups of puppies did become apparent but these were not defined entirely clearly (Table IV).

A change common to all groups was found in sections of the liver in which hæmorrhagic infiltration was manifest by the dilatation of the sinusoids and blood-vessels. This change was most prominent when Canine Virus Hepatitis was present in the dam, with and without the presence of streptococci in the puppy (Groups III and II). Hæmorrhages were found in the group showing streptococci (Group I) and in one case only in the Canine Virus Hepatitis group (Group II). The parenchymal cells were swollen, especially in Groups I and II. Eosinophilic properties in the cytoplasm were most common in Group I. A histiocytic reaction was more common in Group II, as was perivascular cellular infiltration, but the latter also occurred in Groups I and III. There was a partial loss of reticulum pattern in the first and second groups whilst hyaline deposits around the blood-vessels were more common in the second group.

Disappearance of the parenchymal cells was absent in all groups except for small foci in one case from each group where the parenchymal cells had disappeared. Regressive changes in the nuclei of the parenchymal cells were

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seen in all three groups, but these were more prominent in the second and third groups.

Intranuclear inclusion bodies, which are very uncommon in puppies, were found only in the third group in three puppies.

Changes in the lungs were in the form of either inflammation or œdema. The former were manifest by a distension of the blood-vessels and by changes found in the bronchial epithelium, the latter in some cases being desquamated and proliferated. These changes occurred predominantly in Group I. An inflammatory exudate in the lumen of the bronchi was seen in one case in each group. The second type of change was an œdema of the lungs which was more manifest in Groups II and III, and was found in only three cases in Group I. This œdema was not general but appeared in patches.

Changes found in the kidneys were similar in all three groups. They comprised hyperæmia of the cortex, cloudy swelling, fatty infiltration of the convoluted tubules, cellularity and enlargement and adhesions of the glomerular tufts. The convoluted tubules were disorganised and vacuolation and swelling were observed in the tubular linings.

In the spleen there was a similar degree of differentiation among the three groups to that found in the other organs. Œdematous infiltration of the white pulp was seen in all three groups but was most common in Group I. Hæmorrhagic infiltration of the red pulp was found in all groups but was most common in Group II. Disappearance of the white pulp occurred in all groups but was most common in Group I. Regressive changes in the nuclei were present in all groups.

The low percentage of beta-hæmolytic streptococci isolated from the carcases (15.9 per cent) would on first consideration suggest that they are not a significant factor in puppy mortality. Such a conclusion should perhaps be modified, in the light of two conditions which were inevitably imposed on this investigation. The first is that many puppies had been dead for at least 48 hours before being examined and that, although Laughton (1948) found that streptococci on swabs remained viable for at least 24 hours in the post, it may be that the environment in carcase tissue is less favourable to them. If this is so, then it is possible that the percentage present in life was significantly higher than that isolated after death. The second condition is the present widespread use of antibiotics in the field which, although they failed to save the life of the puppy, may exert a bactericidal effect if the tissues are saturated with them or if they are not broken down rapidly.

The figures shown in Table I indicate that in a population of fading puppies, the number which show no evidence of streptococcal infection but whose dams have been infected with the virus of Canine Hepatitis, is approximately equal to that showing streptococci, and that the simultaneous occurrence of the two infective agents is sufficiently uncommon to be regarded as accidental.

Canine Virus Hepatitis is difficult to diagnose with certainty in the young puppy because no clearly defined, specific histological changes occur. Apparently,

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intranuclear inclusion bodies which are believed to occur in the terminal stages of the infection do not form readily in the young puppy, and the complement-fixation test does not show the presence of any antigen in the liver of such young animals. Since the results of tests made for the presence of antigen in the dam's milk were mostly negative, and inconclusive where positive results were obtained, the time and route of infection from bitch to offspring is still in doubt, if it takes place.

No attempt has been made in this study to approach the complicated problem of the possible effect of the absorption of toxins *in utero,* as suggested by Hare and Fry (1938) in streptococcal infections, to account for the death of whelps.

Our final conclusions must take into consideration that in puppies and their dams, infection with beta-hæmolytic streptococci and Canine Hepatitis Virus, either individually or together, accounted for approximately one-third (32.1 per cent) of the mortality among the puppies studied. It must be admitted, therefore, that the fading puppy syndrome is not necessarily caused always by these two infectibe agents, and that other factors must be considered as primary or contributory causes.

Summary

1. The carcases of puppies showing the fading syndrome were examined for beta-hæmolytic streptococci and Canine Hepatitis virus, and for specific histopathological changes.

2. Histological changes alone were not found to be defined sufficiently clearly to allow a definite diagnosis of the infection present to be made.

3. The presence of intranuclear inclusion bodies, which are regarded as specific for hepatitis in the adult dog, were found in three cases only of young puppies.

4. The liver of some of the puppies was tested for antigen of Canine Hepatitis virus, and blood and milk from the dams of these puppies was tested for antibody and antigen.

5. Positive results were not obtained in a sufficiently high proportion of cases to indicate that the two infective agents, beta-hæmolytic streptococci and Canine Hepatitis virus, acting alone or in combination, are a major cause of neo-natal mortality in the puppy.

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THE ANATOMY OF A CASE OF *CEPHALOTHORACOPAGUS MONOSYMMETROS* IN THE BOVINE SPECIES—PART II

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(*Continued from August issue*)

Alimentary Canal

The arrangement of the alimentary canal in *Cephalothoracopagus* shows a remarkable degree of constancy, allowing for species differences, and only rarely is an exceptional case described in the literature. One such exceptional case is mentioned by Rohrbach (1952) in the sheep. The only other detailed description in the ruminant, that by Arndt (1925), concerns a case in the goat, and there is considerable similarity in the disposition of the alimentary canal with that of the present case.

1. Mouth.

The mouth showed the main tongue element lying, as stated, in the wide cleft which replaced the hard and soft palates. This tongue appeared normal and bore the usual relations to the pharyngeal region. Dorsally and posteriorly in the pharyngeal region there was a small prominence, 1 cm. in diameter, covered with papillæ. This was situated below the conjoined temporal regions of the incomplete side of the head, and clearly represented the tongue of this side of the head in a suppressed state of development. As far as can be determined, this is the first time that such tongue development has been recorded.

2. Œsophagus.

The œsophagus, as in all similar recorded cases, was single. It was wider than usual and gave the impression of having developed from both individual elements.

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3. Stomach.

In species where a simple stomach is normally present, this region is represented by a single tube with a flask-like swelling on each side. this representing an incomplete duplication of the stomach. In the present case, as in

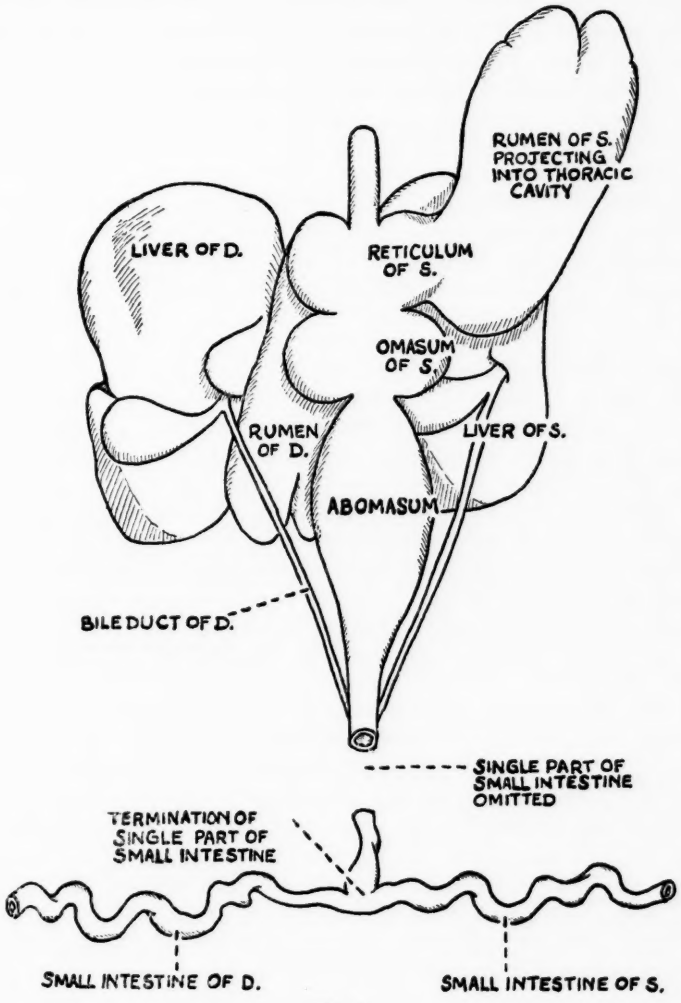
RUMEN OF S. PROJECTING INTO THORACIC CAVITY
LIVER OF D.
RETICULUM OF S.
OMASUM OF S.
RUMEN OF D.
LIVER OF S.
ABOMASUM
BILE DUCT OF D.
SINGLE PART OF SMALL INTESTINE OMITTED
TERMINATION OF SINGLE PART OF SMALL INTESTINE
SMALL INTESTINE OF D.
SMALL INTESTINE OF S.

Fig. 6

that described by Arndt (1925), the œsophagus was continued by a tubular structure which proved to be a common œsophageal (reticular) groove, and which showed two characteristic pillars on each side. These pillars guarded the openings into the two reticula, one on each side, which in turn opened each into

a rumen. Thus the reticula and rumina appeared as outpocketings from the region of the common œsophageal groove. The rumen and reticulum of D were arranged in the usual manner, while those of S retained their primitive forward disposition and protruded through the opening in the diaphragm into the pleural cavity on the left of the ventral sternum. The common œsophageal groove was succeeded by a common single tube carrying an omasum on each side as a dilatation. The tube represented a common sulcus omasi and led backwards to a single, widely-dilated abomasum, which had clearly undergone partial duplication in producing the dilation.

4. Livers and Spleens.

The livers were distinct from one another and were normal in size, shape and position. The presence of a small connection between the two livers has been frequently recorded in these cases. Each spleen was normally developed and was related to the corresponding rumen, that of S projecting through the diaphragm with the rumen.

5. Small and Large Intestines.

Most cases of *Cephalothoracopagus* show a small intestine which is single in the first part of its course and double in the latter part. The site of division into two parts is stated by Schwalbe (1907) and others to be the position of the yolk stalk. The case of Vrolik cited by Schwalbe (1907) is the only one recorded where division took place much further posteriorly (at the point of origin of the cæca). In the present case the length of the single part of the gut was approximately 375 cm., while from the bifurcation to the cæcum the length was 300 cm. in S and 190 cm. in D. Once the cæcum had been reached the intestines were disposed in a completely normal manner, lying in their own posterior abdominal cavities, since at this level the cavities had become distinct from one another.

In connection with the single part of the intestine several points of interest were noted. For the first 4 cm. of its course it ran straight backwards and was then joined by the greatly-elongated bile and pancreatic ducts of the two individual elements. These ducts opened opposite to one another, and the gut appeared to be stretched between the two bile and the two pancreatic ducts. After the entry of these ducts the single intestine followed an extremely convoluted course, being supported on each side by a mesentery, each mesentery arising from the roof of the abdominal cavity of one of the elements, and carrying a series of mesenteric vessels. The convolutions were formed in such a way that a twist was formed first above this mesentery and then below. The intestine lay slightly to the right of the middle line of the whole monster. At its bifurcation a very slight dilatation was formed but this was not so well defined as the pouching which has been described as a common occurrence.

Circulatory System

The description of the circulatory system must precede that of the respiratory system as the relations of the latter are connected so closely with those of the

hearts. The literature on the subject indicates that two hearts are always present in cases in the human subject, but in animals there is frequently only one heart (see particularly Engel, 1931, and Rohrbach, 1952). The present case was one of a small number in which two complete hearts were present. Whenever two hearts occur these are arranged in a typical manner, resulting from the production of two composite hearts in the very early stages of embryonic development. The arrangement has been described on numerous occasions in some detail, although the significance has not always been appreciated (Carey, 1917). At some very early stage in development, presumably at the stage when partial fission has occurred, the precursors of the parts of the two hearts become interchanged so that a dorsal and a ventral heart are formed, related to the dorsal and ventral sterna. The dorsal heart has resulted from the combination of the precursors of the right side of the heart of S and the left side of the heart of D. In a similar manner the ventral heart represents a combination of the left side of the heart of S and the right side of the heart of D. The hearts are normal in appearance, are usually well-formed, but may differ slightly in size. Following on this combination of hearts, there are characteristic arrangements of the associated vessels. Thus, all the veins of individual element S ultimately drain into the dorsal heart which, however, gives off the aorta, and so the arteries, of individual element D. Similarly, the ventral heart receives all the venous blood from D and supplies all the arterial blood to S (Fig. 5). In all cases the aortæ are connected with one another by a short vessel of equal calibre, which Schwalbe (1907) considers to be an additional branchial arch.

This arrangement was present in the case under consideration. It will be realised that this transposition of vessels produces many complexities of relationship and position in the smaller vessels of the thoracic region, but it is felt that for this description only the main differences need be mentioned.

The two hearts had the usual appearance, the dorsal one being slightly larger than that lying ventrally. There were three obvious small defects in the interventricular septum of the dorsal heart, but otherwise the interiors were typical. The foramen ovale of each heart showed indications of commencing closure. The connection between the two arterial systems was formed by a continuity between the two common brachio-cephalic trunks (the anterior aortæ), and the arch so formed gave off the common carotid arteries. The umbilical arteries were two in number in each individual element. The right vessel in S and the left of D were fully developed, while the others were minute vessels, normal in position and reaching to the umbilicus. From the cases which have been recorded in sufficient detail it is clear that such reduction, or even absence, of some of the umbilical arteries is common, but there is no constancy in the disposition.

In the venous system the most marked peculiarity was the presence of a vena azygos (as in the horse and dog) in place of the vena hemiazygos which is the usual bovine arrangement. An umbilical vein ran to each liver.

Each heart showed the usual pulmonary artery and associated ductus arteriosus and the usual pulmonary veins which were connected with the lungs.

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Respiratory System

Four lungs were present, two of which were recognisably left and two were right. These were arranged in two pairs, each pair being related to one of the hearts (and thus to one of the sterna), and being connected by the pulmonary vessels with this heart. These lungs showed no indication of orientation in relation to the vertebral columns. Schwalbe (1907), basing his theory on the report of Rühe, suggests that, as with all the other composite structures, the right lung of D and the left lung of S form the pair on each side of the ventral heart, and this may well be so, but it is quite definite that the vessels connected with this ventral heart in the present case. This could only be explained by a secondary formation of these vessels in relation to the adjacent lungs.

The lungs themselves were normal in appearance and lobation, but the left ventral lung was reduced in size owing to the projection into its pleural cavity of part of the rumen and spleen of S. Apart from the usual mediastina, each pleural sac formed an additional fold with one of the sacs of the other element, which was related to one of the aortæ.

The tracheæ lay dorsal and ventral to the single œsophagus and each showed a normal larynx. These larynges were orientated to the œsophagus in the usual manner so that, in each case, the epiglottis lay on the side away from the œsophagus. Each was suspended from the skull by a hyoid apparatus and the dorsal larynx was slightly the smaller.

Nervous System

In the nervous system duplication involved all structures posterior to the level of the infundibulum of the hypophysis. This corresponds with most of the recorded cases. The cerebral hemispheres formed a single pair throughout their extent and the olfactory bulbs, the optic chiasma, the hypophysis and the infundibulum were all apparently single. Immediately behind the infundibulum there was clear indication of duplication. The cerebral peduncles were involved, as was the mammillary body which was abnormally wide. The posterior parts of the cerebral peduncles were completely double and distinctly separated from one another. More dorsally the epiphysis and thalamus were single, but the corpora quadrigemina were double, although the axial members of each anterior pair lay in close contact. The duplicated parts came into continuity in the region of the duplicated (axial) medial geniculate bodies. As would be expected from the above description, the third ventricle was single but at its posterior extremity it became continuous with two cerebral aqueducts, each beneath one group of corpora quadrigemina. Two distinct cerebella were present.

The first and second cranial nerves were single, but all the remaining cranial nerves were doubled. In common with the distortion and compression of the other structures on the incomplete surface of the head, the axial members of each pair showed varying degrees of development. From the third to the sixth the two axial nerves were very poorly developed, consisting of a very few fine filaments ending in the dura mater, except in the case of the fifth where the fibres ended in the intracranial muscle tissue already described. The axial seventh cranial nerves were fairly large and were apparently concerned with

the supply of the intracranial muscle, as well as the supply of the middle ear, and a large cervical branch was also present. The eighth to the eleventh nerves all appeared to be fully developed and, as far as could be determined, had normal courses and distributions. In the œsophageal region the vagus nerves, axial and abaxial, became united to form two nerve trunks, one dorsal and one ventral

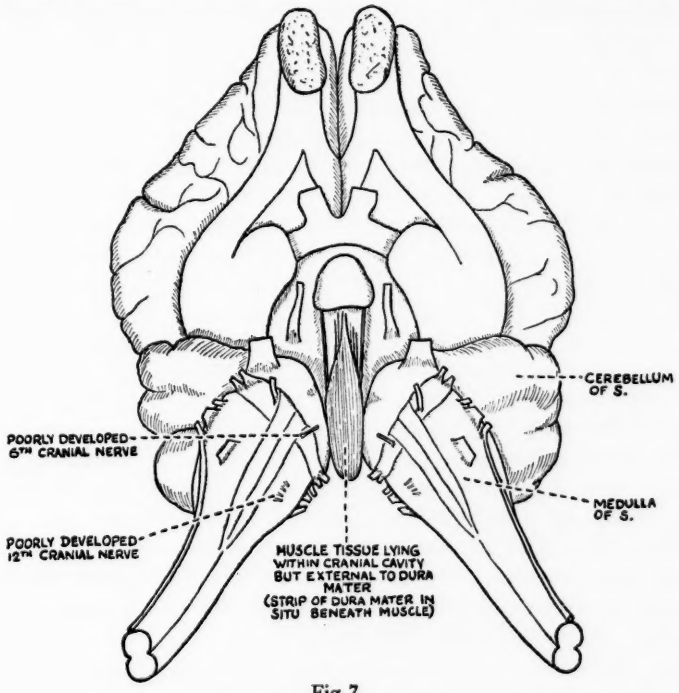
CEREBELLUM OF S.
POORLY DEVELOPED 6TH CRANIAL NERVE
MEDULLA OF S.
POORLY DEVELOPED 12TH CRANIAL NERVE
MUSCLE TISSUE LYING WITHIN CRANIAL CAVITY BUT EXTERNAL TO DURA MATER
(STRIP OF DURA MATER IN SITU BENEATH MUSCLE)

Fig 7
Drawing of base of brain.

to the œsophagus. The dorsal trunk was formed by the right nerve of S and the left nerve of D, and the ventral trunk was formed by the other nerves. On the abdominal viscera the distribution of the branches suggested that the dorsal nerve trunk represented the dorsal œsophageal trunk of S and the ventral trunk of D, this corresponding in part with the usual arrangement, in which the dorsal œsophageal trunk receives most of its fibres from the right vagus. The twelfth cranial nerves were very poorly developed axially corresponding with the poorly-developed tongue on the incomplete surface of the head.

Urogenital System

Two complete sets of urinary organs and two complete sets of female genital organs were present which were normal in every respect.

Discussion

As far as can be seen from the literature there are no recent reports in English on cases of *Cephalothoracopagus* in animals and, as this present case

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concerned a large animal, it was considered worthy of detailed examination and description. In these cases it is surprising how many apparently individual organs are, in fact, composite in formation. The incomplete separation anteriorly produces, as it were, four axes, two of which correspond with the vertebral columns and two correspond with the composite sterna and faces. The tendency for the gut to duplicate appears to be far less than the tendency in other organs, and it is interesting that the bodies only become completely separate behind the level of the umbilicus.

For the formation of the various composite structures it is obvious that the partial separation must take place at a very early stage in development and, according to Schwalbe (1907), this must be before the precursors of the hearts are laid down.

The sex of the monster is of interest. Various estimates are given of the frequency with which this occurs in females compared with males. Where such abnormalities occur rarely and where accounts are often incomplete, it is difficult to be precise, but Arndt (1925) and Rohrbach (1952) suggest that there are three cases in the female to one case in the male. Surveying the whole literature of partial duplication in man and in animals, the general impression is one of a preponderance of females. It is worthy of note that cases of partial duplication in males seem to show additional subsidiary defects more commonly than do females. Williams (1935, 1936) states that a large proportion of aborts and young dead at term are males and he gives details of the non-viability of males as compared with females in young after birth. These facts apply, of course, to the bovine species, but if they are applicable generally and if they are applicable to early embryonic life, it may be that the influence which produces duplication, partial or complete, in some males and females produces death in a larger proportion of males than females.

The suggestion that such conjoined twins could result from the union of two originally separate embryos has long been discarded, but the actual cause of the fission is still a matter of conjecture. Duplication has been caused mechanically, e.g., by Spemann (1938), and chemically, e.g., by Stockard (1921), but these are usually anterior duplications. Wolff (1948) has shown that it is possible to obtain at will almost all the common malformations of the chick embryo by exposing minutely localised regions of the early embryo at specific times to X-rays, but no mention is made of twinning of the embryo. It seems clear that any form of malformation is a result of some external influence at a given time which, according to most authors, has the effect of slowing the development rate. Depending on the time at which the influence acts various malformations result, and it may be noted that the same influence, e.g., vitamin A deficiency, may produce quite different defects in different species (Abercrombie, 1954). It is agreed that the general effects—fission, partial or complete—are produced by influences acting at an earlier stage than those which produce the more localised effects. One question which is still unanswered is whether partially separate twins result from an incomplete fission of an early embryonic stage, or whether fission has been complete but the results of fission

have been so close to one another that coalescence has occurred. Apparently twinning, partial or complete, is an attempt to increase the possibilities of survival in response to the effects of the external influence, and this influence may be the presence of some harmful substance or the absence of some essential substance.

From a practical point of view it is clearly important that such cases should be avoided. Not only is there great danger to the mother when the time for parturition arrives, but there is also the loss of what might have been a healthy normal calf. It would appear to be essential that the cow should be in as perfect a state of health and environment as possible at the time immediately before and after conception if cases are to be avoided. Williams (1931) states that in any country the greatest number of monstrosities occur in dairy cattle as compared with any other species. He considers that the main causes are to be found in genital disease, bad feeding, excessive breeding and milking, and bad management in calfhood.

Summary

Part I of the paper gives a description of the removal of a *Cephalothoracopagus monosymmetros* from a parturient Friesian heifer by Cæsarian section, followed by the uneventful recovery of the mother.

Part II of the paper details the anatomical peculiarities of the monstrosity, comparing it with the previously recorded cases, and commenting on some points of interest in connection with such cases.

Acknowledgments

The authors wish to express their indebtedness to the Heads of their Departments, Professor J. G. Wright, D.Sc., M.V.Sc., F.R.C.V.S., and Mr. H. V. Hughes, B.V.Sc., M.R.C.V.S., D.V.H., for help and encouragement during the course of the preparation of this paper. They are also very grateful to Messrs. B. W. Hayes, B.V.Sc., M.R.C.V.S., and R. B. Taylor, B.V.Sc., M.R.C.V.S., at the time Final Year students, who were responsible for the skilful reassembling of the monster after embryotomy, and to Mr. B. W. Hayes for the excellent photographs.

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ABSTRACT

UNPROTECTED BIRDS, by Edward Morgan, F.R.C.V.S., Venezuela. *Welsh Gazette,* Aberystwyth.

IN a vigorous defence of birds, Mr. Morgan states that starlings and seagulls have been accused of conveying the virus of foot-and-mouth disease from the Continent of Europe into Britain. He has held a government appointment in Venezuela for many years and he has been able to study the problem as to whether birds carry the virus into clean countries from infected lands.

Until about four years ago there were only two countries in S. America which had been fortunate enough to escape foot-and-mouth disease. They were Venezuela and Colombia. Venezuela's frontier borders on to Brazil but vast areas of jungle, deep valleys and wide rivers have helped to prevent contact between the cattle of the two countries. These things do not prevent birds from crossing the frontiers and often in great flocks like the starlings.

Many of the birds are carnivorous and when an animal dies they arrive on the scene in great numbers. In a short time only the bones remain. The carrion birds are black in colour and a little larger than a carrion crow. Locally they are called "Zamuro" which in English would be "Turkey Buzzard." They are protected by the State for they act as scavengers.

They have never brought foot-and-mouth disease to Venezuela from Brazil. About four years ago the disease appeared for the first time in Venezuela. It came to the farm at which the author was writing. The cattle on the neighbouring farm escaped the disease for there is a deep valley with steep cliffs between the two farms and the animals could not come in contact. This limitation does not apply to the birds. They rest in the cliffs and rocks and they are constantly going to and fro from one farm to the other.

Many suckling calves on the author's farm became infected yet his neighbour's cattle remained free of the disease.

When foot-and-mouth disease invaded Mexico the U.S. authorities built a double fence between the two countries. It extended from coast to coast. If birds were carriers then all this effort would have been in vain. The fence proved to be very effective.

The Americans ridicule the idea that birds carry the virus over land and sea. It is "ships" which carry the virus. The disease persists in the Argentine despite 40 years of vaccination. The U.S. authorities prohibit the importation of cattle, sheep, goats and pigs and meat, except canned meat, from S. America.

Five years ago the Venezuelian Food Controller decided to import chilled meat from the Argentine for the first time in history. The Minister of Agriculture, the landowners and the Colombian Government protested because of the danger of importing foot-and-mouth disease. After the meat arrived it was distributed into the country districts and the farmers had a new disease to contend with, i.e., foot-and-mouth disease. Mr. Morgan goes on to enter a plea in the defence of birds. D.J.A.

REVIEWS

THE PHYSIOLOGY OF DOMESTIC ANIMALS, by H. H. Dukes, 1915. Seventh Edition. London: Baillière, Tindall & Cox. Price 75s.

THIS seventh edition of H. H. Dukes's "The Physiology of Domestic Animals" comes eight years after the previous edition and 22 years after the first. The book now contains more than 1,000 pages and 238 illustrations.

In style the new edition remains faithful to the pattern that is known so well by veterinary students. The text shows evidence of considerable revision and there are many additions. Professor Dukes continues to enjoy the collaboration of Dr. Hewitt, who writes on the physicochemical basis of physiological phenomena, Dr. Sumner on physiological oxidations and Dr. Asdell on reproduction, growth and the endocrines. In this edition Dr. Dye contributes a chapter on intermediary metabolism and Dr. Kare on water, electrolytes and acid-base balance. The new references at the end of most chapters are kept as a separate list and the title of the article is given in full. Professor Dukes has been quite eclectic in selecting his new sources.

A new feature of this edition is an appendix entitled "Lecture demonstrations in physiology." Here are listed some 112 possible demonstrations covering most organ systems. These demonstrations have been developed by Professor Dukes over a number of years and they are an example of American teaching methods. Demonstrations somewhat similar to those illustrated already play a part in the curricula of most veterinary schools in this country.

The re-writing and revision of this edition will serve to maintain the popularity of "Dukes" which will continue to provide for veterinary students in the same fashion as previous editions. The only feature which will not commend itself to students is, unfortunately, its high price.

A TEXTBOOK OF PHYSIOLOGY, by John F. Fulton. 17th Edition. 1955. London: W. B. Saunders Company. Price, 94s. 6d.

THE 17th edition of this book has now become a Textbook of Physiology, edited by Professor J. F. Fulton. It was originally known as Howells' Textbook of Physiology.

Professor Fulton continues to enjoy the collaboration of many of the most eminent physiologists, biochemists and anatomists in the United States as well as two from this country. Although these experts cover their own specialities in various sections and chapters, the book is not uneven which is indicative of good editing.

The whole of the new text is most up to date and, in order to be so, it has been extensively rewritten. This is most apparent in the chapters which deal with those matters which are showing the most rapid development. This revision is most evident in the sections covering the principles of nervous activity, respiration, body fluid and kidney function. Professor D. Nachmansohn contributes a chapter in which he propounds his views on acetycholine and energy transformation in nerve cells.

The new edition is a worthy successor of previous editions. It can be

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recommended with confidence as a good textbook of advanced physiology. The book is produced in an excellent manner with a type face to which we have grown accustomed. The documentation and index are of the very high standard always set by Professor Fulton.

ANIMAL NUTRITION, by Maynard and Loosli. Fourth edition. McGraw-Hill Book Company, Inc. Price, 56s.

ALTHOUGH the pattern of this book has remained essentially the same since its first edition published in 1937 its contents have been revised extensively and brought up to date. In the five years since the last edition considerable progress has been made in nutritional research and this is reflected in an entirely new chapter on "Antibiotics, Hormones, and other Growth-stimulating Substances" which gives a critical appraisal of present knowledge. New techniques for evaluating the digestibility of feeds by using markers in the feed such as chromic oxide, chromagens and radio active isotopes are discussed fully. New developments in the amino acid, vitamin and mineral fields are included in this edition and as usual the text is documented with references to the literature and guides to further reading.

The illustrations, tables and presentation are of a high standard and this book maintains the place gained by previous editions for a textbook on the fundamentals of animal nutrition which has no equal.

ADVANCES IN VETERINARY SCIENCE, Vol. II. Editors: C. A. Brandly and E. L. Jungherr. New York: Academic Press, Inc. Price, $10.00.

THE sum of knowledge is added to at such a great rate and recorded in such a spate of words that it is impossible for the ordinary man to keep pace. It is good, therefore, when those who are well informed on the latest developments in any particular sphere put pen to paper and provide a summary of the latest relevant information.

Advances in Veterinary Science, Volume II, is a collection of such articles. Here are the contents:

1. Epizootiology of Virus Diseases. Richard & Shope.
2. Mycosis of Animals. Chester W. Emmons.
3. Respiratory Diseases of Poultry. Henry Van Rockel.
4. The Blood Groups of Animals. L. C. Ferguson.
5. Diseases caused by Deficience of Trace Elements. I. J. Cunningham.
6. Photo Sensitisation in Animals. N. T. Clark.
7. Rumen Dysfunction. A. T. Phillipson.
8. Bovine Ketosis. J. C. Shaw.
9. Tick-borne Rickettsiosis in South Africa. D. A. Haig.
10. Vibriosis. W. N. Plastridge.
11. Effective Control of Internal Parasites. Donald E. Boughton.

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The reader is presented with the latest information by competent authorities. Each article is supported by an extensive bibliography which, in every case, is a witness of the tremendous amount of work accomplished and in progress and also of the value of a single article summarising all the essential and valuable information.

NOTES AND NEWS

Inter-African Advisory Committee for Epizootic Diseases Third Annual Meeting

THE above C.C.T.A. meeting under the chairmanship of Mr. R. S. Marshall, C.B.E., Veterinary Adviser to the U.K. Colonial Office, was held in Pretoria from July 2 to 4, 1956. Veterinary representatives from each off the six member countries of the Commission, namely, Belgium, France, Federation of Rhodesia and Nyasaland, Portugal, Union of South Africa and the U.K., were present.

Technical reports were received on the following subjects:

1. Murine adapted strains of asterococcus mycoides in the immunisation of cattle against bovine pleuropneumonia, by Dr. Gerlach.—Angola.

2. A quantitative complement-fixation test for contagious bovine pleuropneumonia, by Mr. R. C. Newing.—Kenya.

3. Recent advances in rinderpest vaccine research, by Dr. S. E. Piercy.—Kenya.

4. A review of the bovine theilerioses, by Dr. W. O. Neitz.—Union of South Africa.

5. Prophylaxis against rabies in Southern Africa by D. A. Lawrence.—Federation of Rhodesia and Nyasaland.

Further consideration was given to inter-territorial projects including rinderpest, survey of wild life, a statistical survey of clinical and laboratory data in rabies, regulations for the exchange of virulent material and the establishment of regional committee on epizootic diseases.

It was resolved that an international convention for the control of rinderpest be drafted and a specialist meeting of experts on laboratory techniques in contagious bovine pleuropneumonia be convened.

The technical reports for the 1957 meeting will fall under the title of " Important Virus Diseases of Animals transmitted by Arthropoda."

An invitation to hold the fourth annual meeting at Dakar was received from the French Government, and Dr. D. A. Lawrence, Director of Veterinary Services, Southern Rhodesia, was elected chairman for 1956-57.

The Feeding of Milk to Calves in Transit

THE Minister of Agriculture, Fisheries and Food and the Secretary of State for Scotland have made an Order prohibiting the feeding of milk to calves in transit, except milk which has been reconstituted from dried milk. The purpose of this regulation is to safeguard against the risk of spreading foot-and-mouth disease arising from feeding calves with raw milk which might have been infected.

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Attention was drawn to this risk by the Gowers Committee on Foot-and-Mouth Disease.

Copies of the Order, entitled The Calves (Feeding in Transit) Order, 1956 (S.I.1956/1127), may be obtained from H.M. Stationery Office, price 3d. net.

BURROUGHS WELLCOME & Co announce the introduction of "Hepadis" brand Distemper, Hard Pad and Contagious Hepatitis Antiserum.

This antiserum is prepared in dogs hyperimmunised against the viruses of distemper, hard pad disease and contagious hepatitis and also injected with mixed bacterial antigens. When used prophylactically, a dose of 5 c.c. is given subcutaneously, irrespective of the age or size of the dog. For therapeutic purposes, the antiserum is best given early in the course of the disease, in doses of 0.5 c.c. per lb. bodyweight.

It is issued in containers of 5 c.c. and 25 c.c.

ALLEN & HANBURYS LTD. introduce "Dequadin Wound Powder, which is an effective prophylactic and therapeutic topical dressing for veterinary use in both clean and contaminated wounds."

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Dequadin Wound Powder is issued in packs for dispensing and is available in cartons containing one dozen 15 g. screw-capped tubes and in 200 g. aluminium containers. Prices as follows: Dequadin Wound Powder containing 0.1 per cent Dequadin in a specially compounded urea base. Professional. Cartons of 1 dozen 15-g. tubes, 11s. 8d. each. Container of 200 g., 7s. 5d. each. Exempt from purchase tax.

ERRATUM

Reference to article by B. S. Rajya, on page 188, May, 1956. Line 11 should read "infra-temporal fossa (Plate I) where it . . ." Page 189, line 14, "The ciliary arteries, unlike those of the buffalo, arise . . ." Plate III, identification mark 8, should be "Arteria retis mirabilis" instead of "Arteria tetis mirabilis."

Publishers' Notices

THE BRITISH VETERINARY JOURNAL, with which is incorporated THE VETERINARY JOURNAL, is published monthly, and copy for advertisements should be in the hands of the advertisement manager not later than the 20th of the preceding month, if proof is required. Tel.: Temple Bar 3386.

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